CARDIOGRAPHY

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CARDIOGRAPHY

BY

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M D D Sc F R C P

PHYSICIAN TO THE CARDIAC DEPARTMENT OF THE LONDON HOSPITAL PHYSICIAN TO OUT PATIENTS OF THE NATIONAL HEART HOSPITAL CONSULTING CARDIOLOGIST TO THE ROYAL NAVY

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TO MY WIFE

whose help made this work possible

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PREFACE

THIS book deals with clinical electrocardiography and phonocardiography, radiography of the heart is by itself such a specialized subject that it would be inappropriate to regard it as falling within the scope of instrumental recording of the heart's movements suggested by the title, cardiography

During the second World War *A Student's Handbook of Clinical Electrocardiography* was allowed to go out of print, and the present manual is to replace it More cardiograms have been added including those taken by chest leads The recognition of added heart sounds and the more precise interpretation of murmurs have each been aided so materially by sound records that a part dealing with phonocardiography has been introduced

Like the previous book, the purpose of the present one is the same, namely, to help the student preparing for a qualifying or higher examination in medicine, and especially to assist hospital medical officers called upon to report on occasional cardiograms, it should also guide medical practitioners to understand the findings communicated to them in patients sent for an examination of the heart

Experimental details have been omitted except where they have been necessary to explain or emphasize certain clinical findings

A particular feature of the older book has been preserved in the standard form of the legend which describes each electrocardiogram, adherence to this plan facilitates the correct interpretation of any tracing

I wish to acknowledge my great indebtedness to Sir John Parkinson, Physician to the Cardiac Department of the London Hospital, for it is to him I owe my early training in cardiology, in later years, too, I have continued to benefit from his advice in the clinical and graphic interpretation of heart disease

Mr William Dicks, Chief Technician to the Department, has collaborated in producing most of the records shown here

WILLIAM EVANS

LONDON, *January*, 1948

PART I

ELECTROCARDIOGRAPHY

INTRODUCTORY

ELECTROCARDIOGRAPHY provides a valuable aid in the recognition of many cardiovascular disorders, especially coronary disease, conducting bundle branch injury, pericardial disease, and in the interpretation of an obscure arrhythmia The electrocardiogram should never be used by itself to decide diagnosis or prognosis, and any abnormality it may show is to be examined alongside the clinical findings Several types of electrocardiographs are now in use they are described fully in the instruction booklets issued by the firms which supply the respective instruments, and the technique of recording electrocardiograms is best acquired from a study of these manuals

The cardiographic leads.—The two electrodes taking off the electric current consequent on cardiac action may be placed at different points of the body Convention has acknowledged the value of three limb leads, but agreement on the best chest leads to use is not yet universal Many such leads are employed in research, but only a small number have so far proved their worth in clinical cardiology, and three (Table I and Fig 1) are used here, namely CR_1 CR_4 (or IVR) and CR_7 In the abbreviated designation of chest leads the letter C

TABLE I

Showing the position of the electrodes in the limb and the three chest leads described in this book

Designation of leads		*Position of electrodes*	
		Indifferent electrode	*Exploring electrode*
Limb	I	Right arm	Left arm
,,	II	Right arm	Left leg
,,	III	Left arm	Left leg
Right pectoral	CR_1	Right arm	At right border of sternum in fourth intercostal space
Apical	CR_4	Right arm	Outer side of apex beat
	IVR	Right arm	Outer side of displaced apex beat
Posterior axillary	CR_7	Right arm	Left posterior axillary line at level of apex beat

stands for chest, R for right arm to which is attached the indifferent electrode, and the sub-numerals indicate the station for the exploring electrode; station 1 is at the right border of the sternum in the fourth intercostal space (CR_1); station 2 is in the same space at the left sternal border (CR_2); station 3 is in the fifth intercostal space midway between the left sternal border and the mammary line (CR_3); station 4 is at the apex beat in the left mammary line (CR_4); stations 5, 6 and 7 are in the anterior, middle, and posterior axilla respectively (CR_5, CR_6 and CR_7). In lead IVR the indifferent electrode is on the right arm and

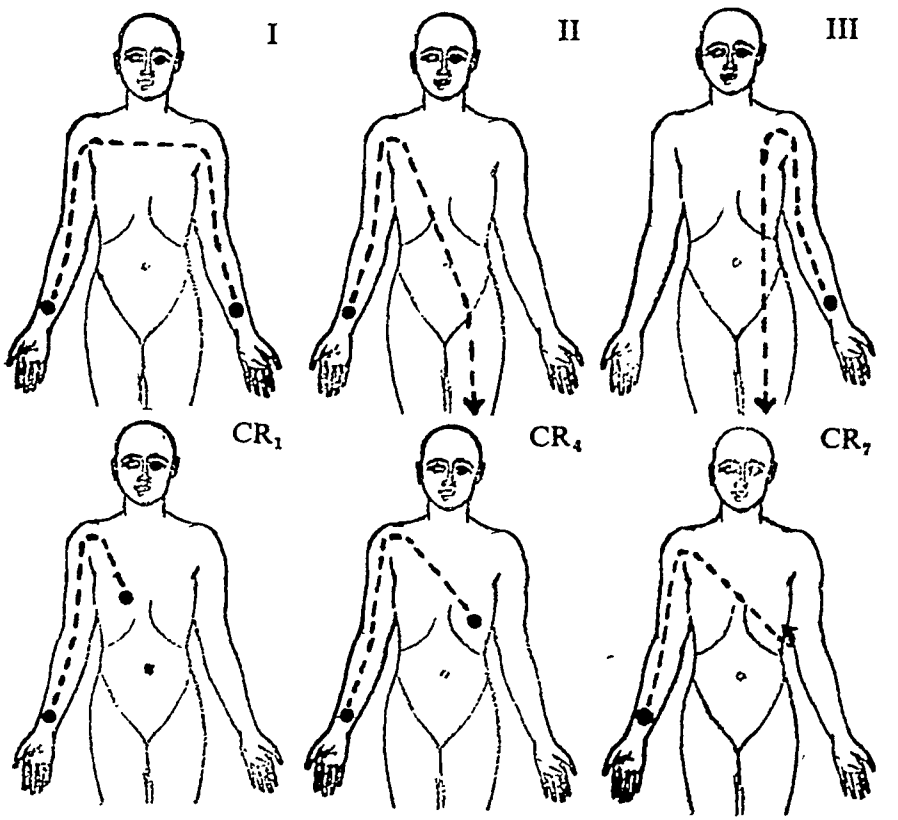


FIG. 1.—Diagram to show how the electrodes are connected in the leads used for cardiograms described in this book.

the exploring electrode over the apex beat whether in its normal position (CR_4) or displaced outwards from enlargement of the heart (IVR).

The chest lead CR_1 should be used whenever failure of the right heart is in doubt, for demonstrating auricular activity, and in infarction of the septum; lead CR_4 or IVR helps in the diagnosis of anterior cardiac infarction, and CR_7 assists in the differential diagnosis of the cardiographic changes in hypertension, posterior cardiac infarction, and right heart preponderance.

The usefulness of vector or unipolar leads is being tested, and they are not mentioned again in this book. In my experience the V leads are not superior to

CR leads in clinical diagnosis CF leads are less useful than CR leads because inversion of the T wave in CF_1, CF_6 and CF_7 is so common in health

HOW TO READ AN ELECTROCARDIOGRAM

Assuming that the tracing has been recorded it is necessary to consider its interpretation The written waves have been conventionally designated P, Q R, S, T and U, and they represent the electrical changes taking place during the progress of cardiac contraction The P wave represents the electrical force produced by activation of the auricular muscle The QRS and T waves represent the electrical force generated when the ventricular muscle contracts and relaxes The U wave is less well understood, but it probably represents some sort of readjustment of the electrical potential within the ventricle The individual components of the QRS complex vary in number from subject to subject and from lead to lead The earliest QRS deflection which lies above the isoelectric level should be labelled R Any downward deflection which precedes R so defined, should be labelled Q The first downward deflection following R should be labelled S The first upward deflection which may follow S should be labelled R and if any downward deflection follows R' it is designated S The term 'diphasic T wave' is applied to a double deflection of the T, one on each side of the isoelectric level, if the first deflection lies below the level the diphasic T is of the minus plus (∓) type or if the reverse applies it is of the plus minus (±) type

In the actual reading of the cardiogram attention should be paid in each lead to some six particulars, and a methodical analysis of these will show how the record differs from the normal, supplying a diagnosis of the condition The following are the six criteria and each in turn should receive separate consideration

1 The rate
2 The rhythm
3 The length of the P–R period
4 The direction of the Electrical Axis
5 The direction amplitude and form of the P wave, QRS complex and the T wave
6 The form of the R–T segment

Thus, whenever an electrocardiogram, in which the leads have been suitably designated, is placed before a student at his examination he should proceed immediately to describe the tracing in accordance with the scheme adopted here He should then summarize the positive findings and pronounce the diagnosis

The importance of adhering to this methodical analysis of the electrocardiogram is emphasized throughout the book because the legend appended to each tracing is described in the same standard way

Before examining the normal or physiological electrocardiogram it is necessary to allude briefly to the way in which the rate is determined, and to describe electrical axis deviation at greater length

How to determine the rate—In order to calculate the duration of any phase of the cardiac cycle depicted in the cardiogram, it is necessary to adopt some means of estimating the speed of the travelling photographic plate or film

Different devices are used for this purpose. A small motor, controlled by a vibrating tuning fork and driving a rotary wheel which carries a spoke, is in common use. As the wheel rotates the spoke interrupts the beam of light so that the record shows a series of lines at regular intervals. It is usual for such intervals to measure $\frac{1}{5}$ second, and four thinner spokes may be arranged to provide intervals of $\frac{1}{25}$ second. Such a device ensures that, although the speed of the film may vary, the rate is shown (Fig. 2).

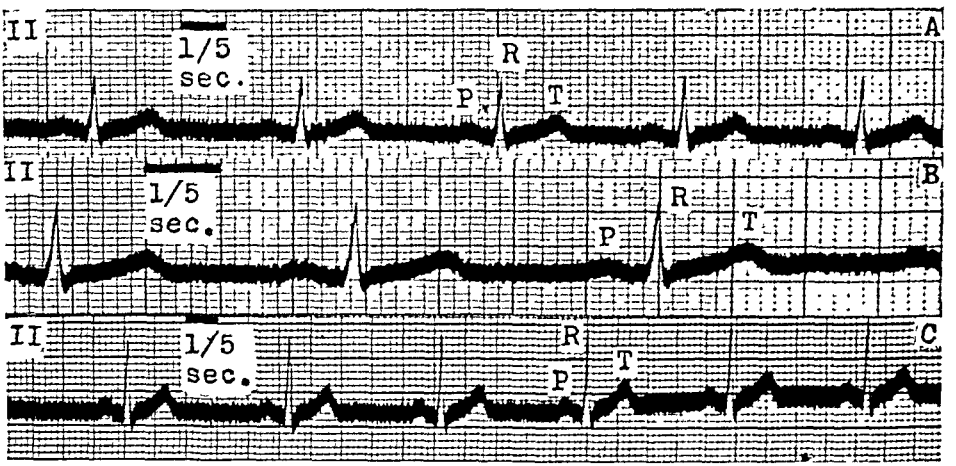


FIG. 2.—Design of the time-marker when the film travels at different speeds. The speed of the film is moderate in (A), rapid in (B), and slow in (C).

TABLE II

Showing heart rate per minute calculated from the number of spaces ($\frac{1}{5}$ second) separating each heart beat.

Number of spaces ($\frac{1}{5}$ second) separating beats	*Calculated rate per minute*	*Number of spaces ($\frac{1}{5}$ second) separating beats*	*Calculated rate per minute*
1	300	6.5	46
1.5	200	7	43
2	150	7.5	40
2.5	120	8	37
3	100	8.5	35
3.5	85	9	33
4	75	9.5	31
4.5	66	10	30
5	60	10.5	29
5.5	55	11	27
6	50	11.5	26

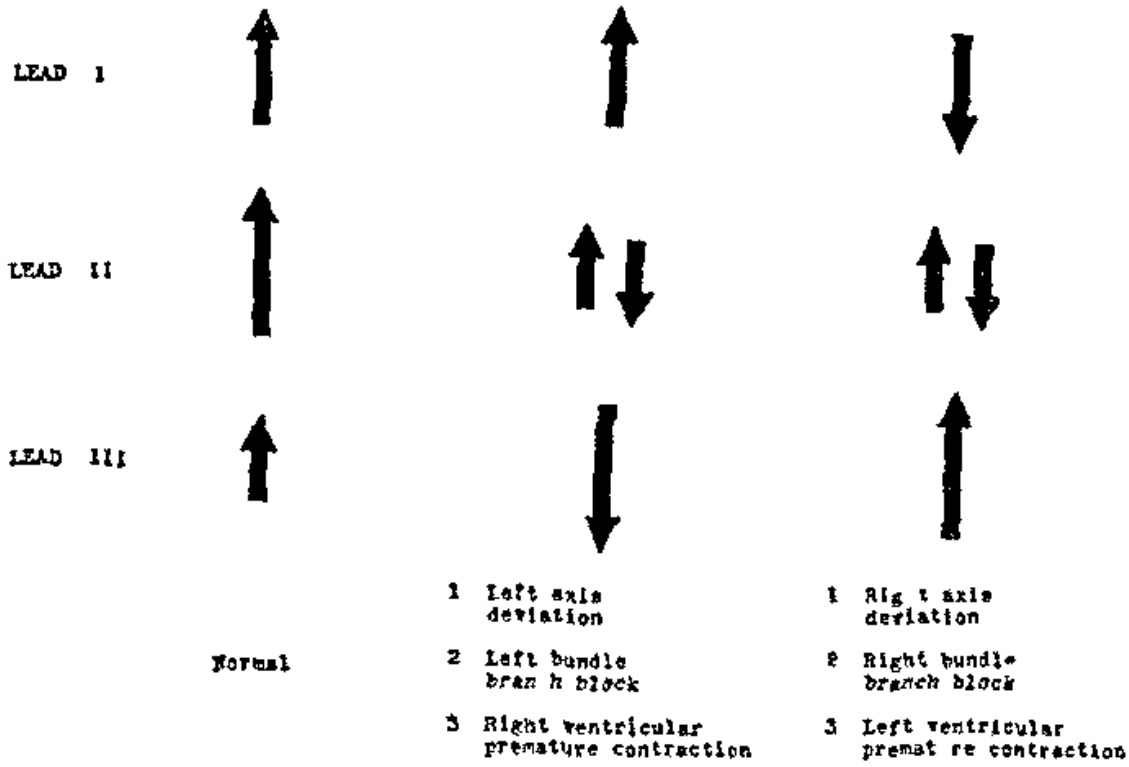


FIG 3.—Diagram to show the direction of the QRS complexes in left (L A D) and right axis deviation (R A D)

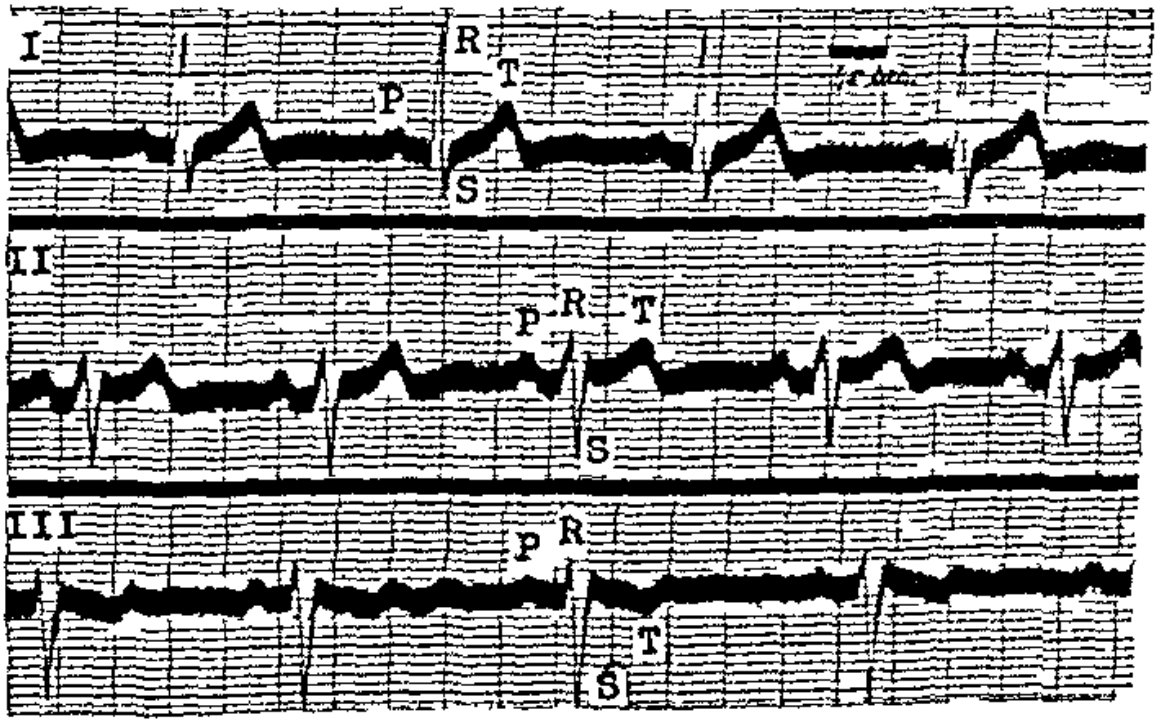


FIG 4.—The rate is normal. The rhythm is regular. The P R period is normal. L A D. The T wave is inverted in lead III

Since the time interval depicted by each space is known, it is possible to estimate the heart rate per minute as well as the duration of any phase of the cardiac cycle. Table II shows the heart rate per minute determined by the number of spaces between heart beats.

Electrical axis deviation.—When the excitation wave spreads through the heart it is attended by a wave of electrical activity which takes a complicated

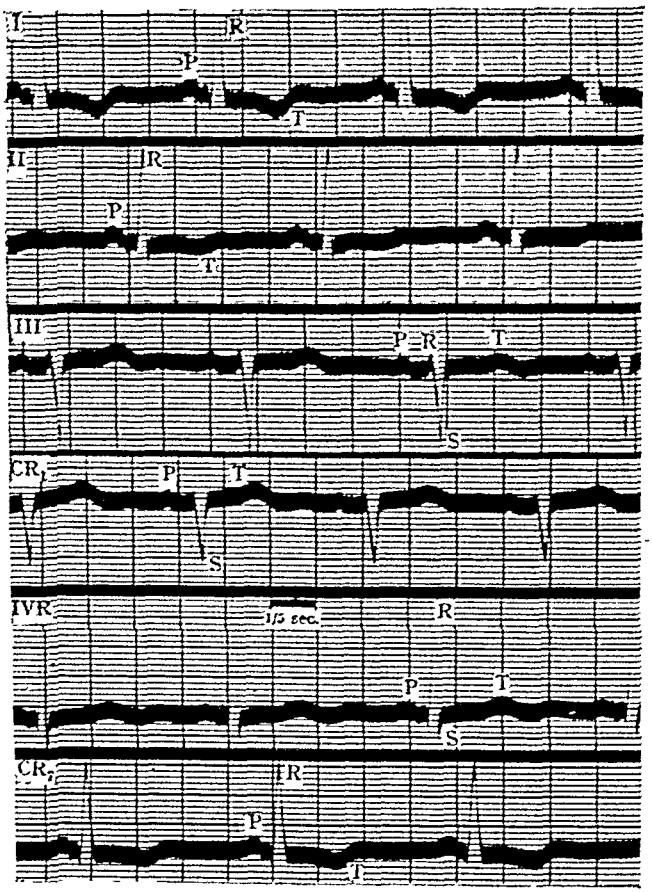


FIG. 5.—The rate is normal. The rhythm is regular. The P–R period is normal. L.A.D. The T wave is inverted in leads I and CR_7, and is diphasic (∓) in lead II. The R wave is low in CR_1.

path. Its course may be represented by an axis, the projection of which is determined in relation to a triangle constructed by the orthodox limb leads of the electrocardiogram. The resultant of this projection of the axis of the distributed electrical potential from the heart contraction is the electrical axis of the electrocardiogram.

If this electrical axis is not deviated from the normal, the main deflection of the QRS complex is upright in each of the three limb leads (Fig. 3).

Left electrical axis deviation—When the main deflection of the QRS complex in lead III is downward and the R wave in lead I is taller than the R wave in

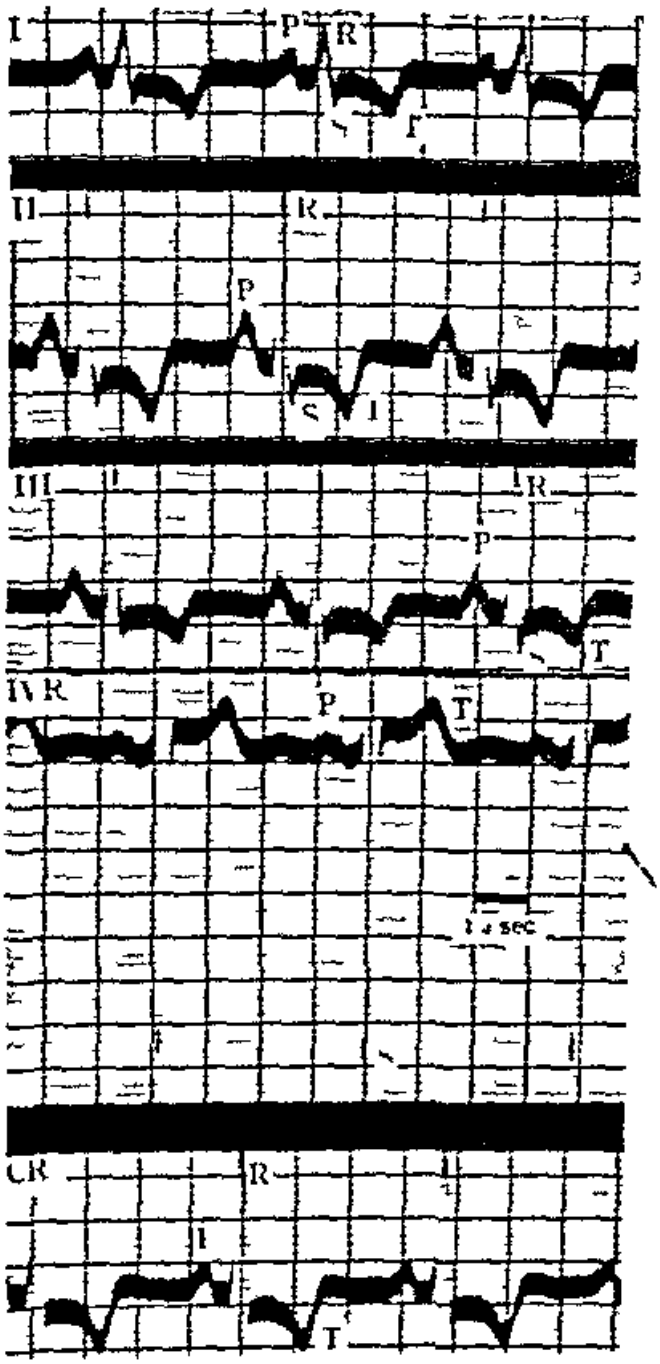


FIG 6—The rate is normal The rhythm is regular The P R period is normal No axis deviation The T wave is inverted in leads I II III and CR, From a case of aortic incompetence without coronary disease (P M control)

lead II, the electrical axis is said to be deviated to the left (Fig 4) Such deviation is present in left bundle branch block

Left axis deviation (L A D) so defined is not a certain sign of preponderant enlargement of the left ventricle although the finding is common in those

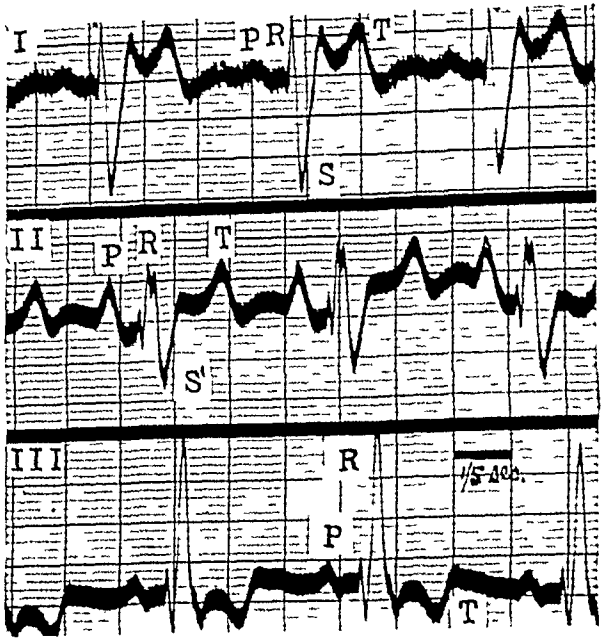


FIG. 7.—The rate is normal. The rhythm is regular The P–R period is normal. R A.D. The P wave is tall and spiky in lead II. The QRS is wide. The T wave is inverted in lead III

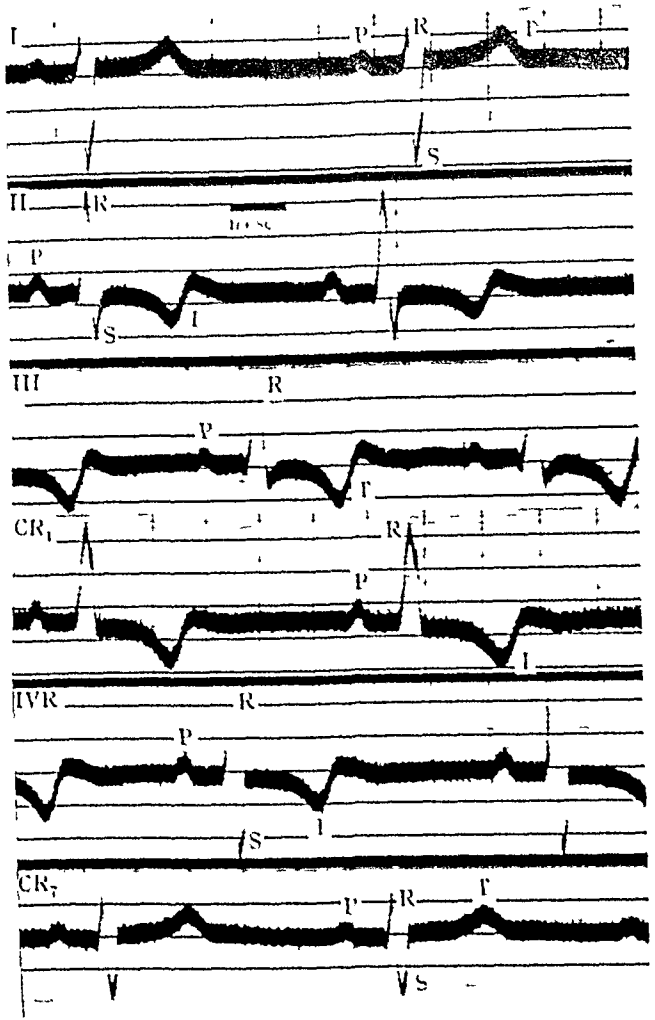


FIG. 8.—The rate is slow from sinus bradycardia. The rhythm is regular. The P–R period is full R.A.D. The T wave is inverted in leads II, III, CR_1 and IVR; it is upright in CR_7. From a case of pulmonary stenosis.

conditions showing left ventricular hypertrophy such as hypertension and aortic incompetence The more dependable cardiographic signs of left ventricular preponderance consist of inversion of the T wave in leads I and CR_7 (Figs 5 and 6) the T in IVR is expected to be upright, but should it be inverted the inversion is less prominent than that in CR_7

Right electrical axis deviation —When the main deflection of the QRS complex in lead I is downward and the R wave in lead III is taller than the R wave in lead II, the electrical axis is said to be deviated to the right (Fig 7) Such deviation is present in right bundle branch block

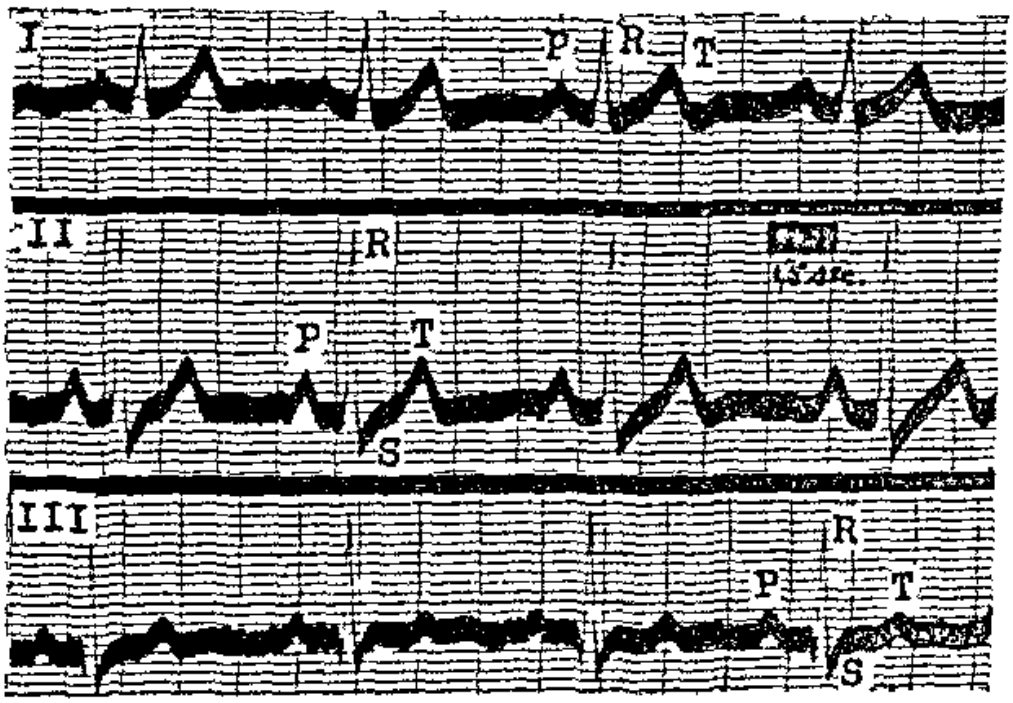


FIG 9 —The rate is normal The rhythm is regular The P R period is normal No electrical axis deviation The P and T waves are normal No abnormal deviation of the R T segments

Right axis deviation (R A D) so defined is not a certain sign of preponderant enlargement of the right heart, although the finding is common in those conditions showing hypertrophy of the right ventricle such as mitral stenosis and congenital heart disease The more dependable signs of right heart preponderance consist of inversion of the T wave in leads II III and in CR_1 the S wave is often absent in CR_1 and prominent in CR_7 (Fig 8)

THE NORMAL OR PHYSIOLOGICAL ELECTROCARDIOGRAM

Physiological electrocardiograms (Figs 9, 10 and 11) from healthy subjects are not identical tracings, for while conforming to a normal pattern they sometimes assume an irregularity which in certain circumstances might be regarded as a departure from the normal

When the features of the normal cardiogram are described in relation to the

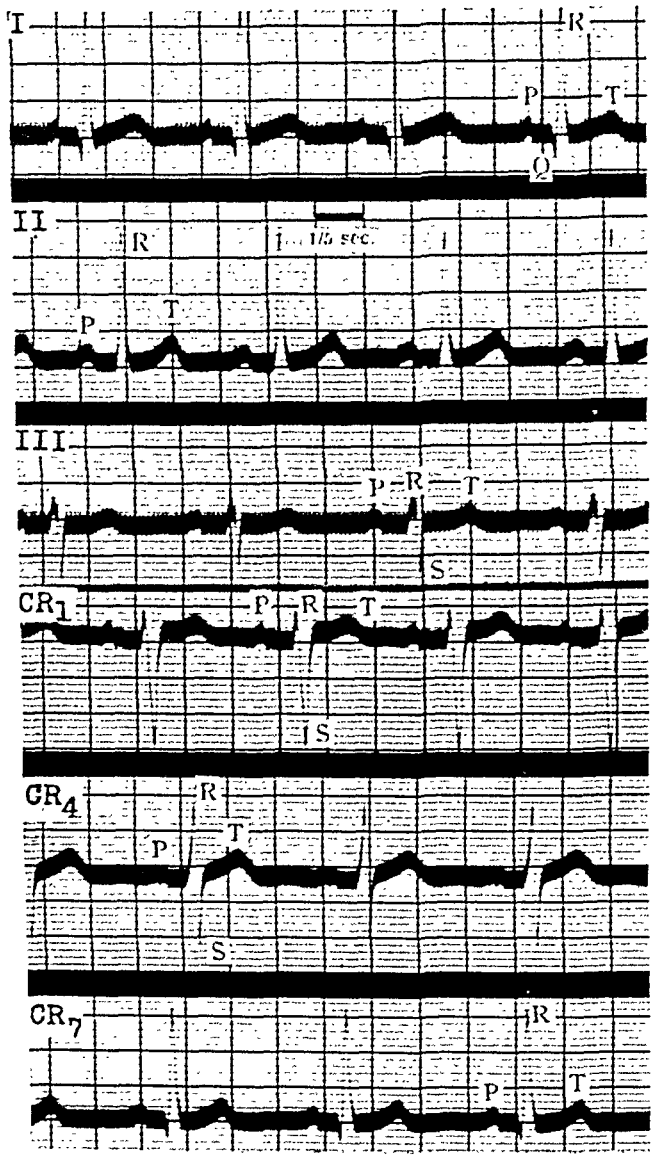


FIG. 10.—The rate is moderate. The rhythm is regular. The P–R period is normal. No electrical axis deviation. The S wave is a little deep in lead III. The P wave is low. The T wave is upright in all leads. From a healthy subject aged 22.

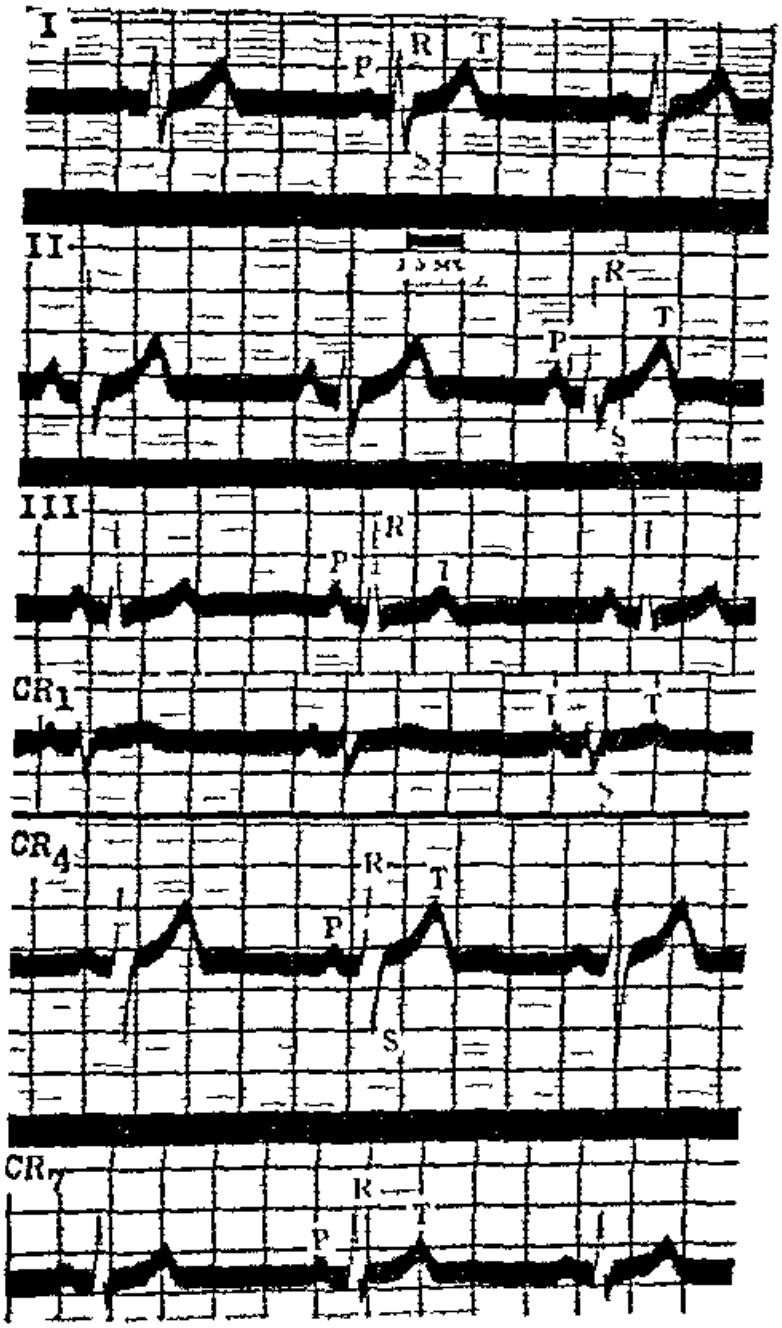


FIG 11 —The rate is normal The rhythm is regular The P R period is normal No electrical axis deviation The three primary waves are normal in all leads From a healthy subject aged 26

six characteristics selected for the methodical analysis of all tracings, they should conform to the following requirements.

1. The rate is neither too slow nor too rapid (60 to 100 a minute).
2. The rhythm is normal, the impulse arising in the sino-auricular node (sinus rhythm).
3. The P–R period (measured from the start of the P wave to the start of the R wave) is neither too short nor prolonged (0·10 to 0·22 second).
4. There is no abnormal deviation of the electrical axis.
5. The P wave, QRS complex, and the T wave, are upright in the limb leads, and of normal amplitude and form. In CR_1 there is never a Q wave and the S wave is greater than the R wave, and in young subjects the T wave may

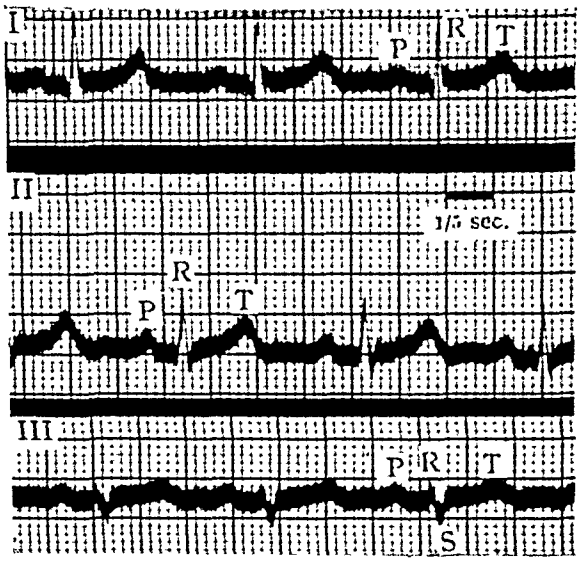


FIG. 12.—The rate is moderate. The rhythm is normal. The P–R period is not abnormal. No abnormal deviation of the electrical axis. The P and T waves are upright. The S–T segment is raised but to an extent less than 1 mm. The tracing is from a healthy youth.

be inverted ; in CR_4 or IVR the S wave may be deep ; in CR_7 the S wave is small.

6. There is neither undue elevation nor depression (greater than 1 millimetre) of the R–T segment.

Even when the above characteristics have been described for the physiological electrocardiogram, variations from the standard tracing might occur without incriminating their owners with any sinister implication. Amongst these are included small, wide or even inverted P waves, widened and splintered QRS complexes, deep S wave, especially in lead I or III, a raising of the S–T segment (Fig. 12), and inversion of the T wave in lead III, and in CR_1 in children.

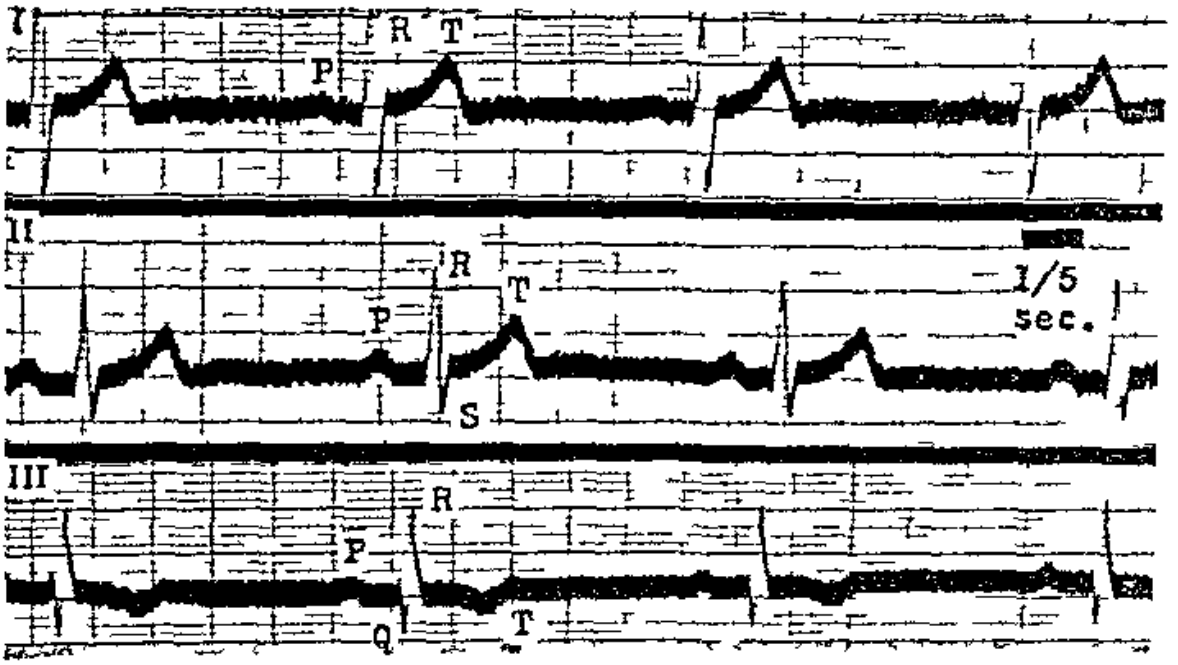


FIG 13.—The rate is slow. The rhythm is regular. The P R period is full. No axis deviation. The Q wave is prominent in lead III, the S wave is deep in lead II, and the T wave is inverted in lead III.

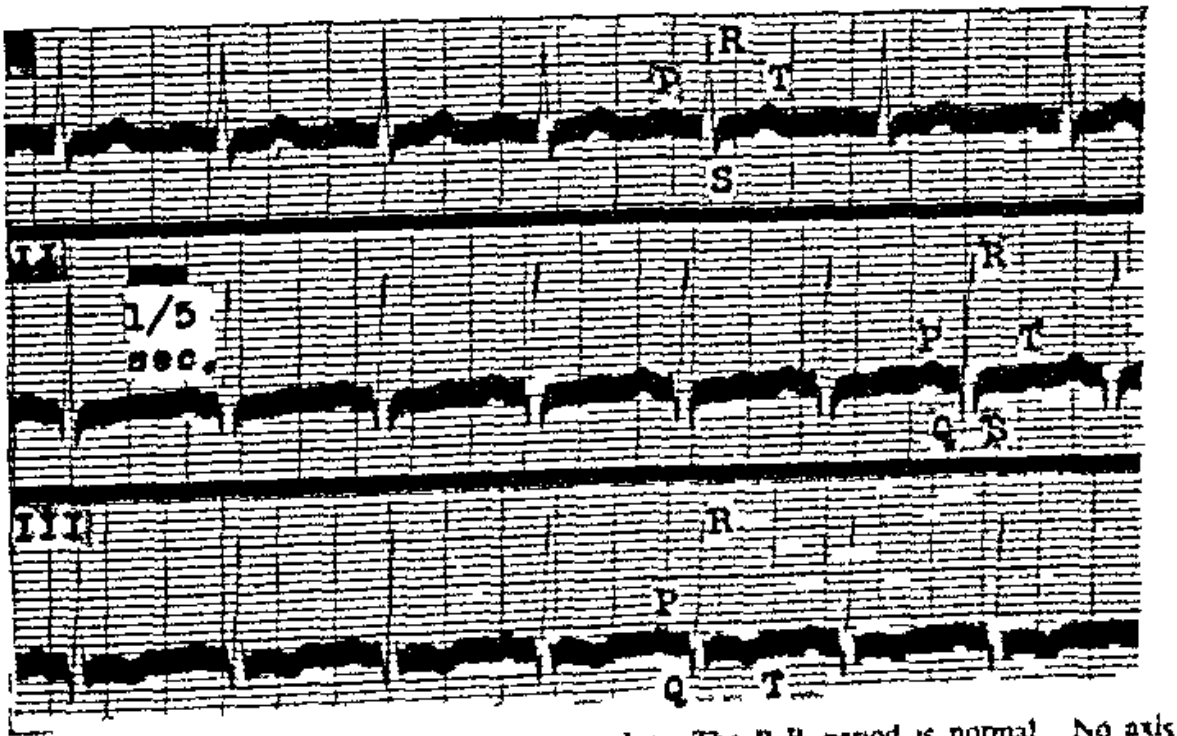


FIG 14.—The rate is rapid. The rhythm is regular. The P R period is normal. No axis deviation. The T wave is low in lead II and inverted in lead III.

ALTERED RHYTHM

ALTERED VAGAL OR SYMPATHETIC INFLUENCE

Sinus bradycardia

In the electrocardiogram of sinus bradycardia (Fig. 13) the P wave conforms to the normal type, and it recurs at regular intervals except in the rare examples of ventricular escape. It is followed by a ventricular complex which need not be abnormal. Similarly the P–R period is not necessarily altered (although it is often prolonged), so that the stimulus commencing in the sinus node is conducted to the ventricles in the usual way. Thus, auriculoventricular systole is normal, but the diastolic period is prolonged. The tracing is only abnormal in that the heart rate is slow (less than 60 a minute); it may be as slow as 50 or even slower.

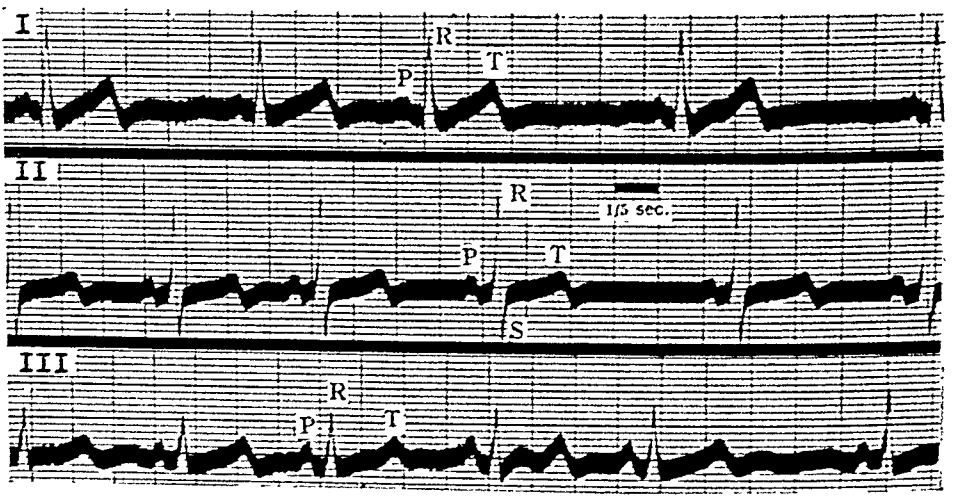


FIG. 15.—The rate is normal. The rhythm is irregular from sinus arrhythmia. The P–R period is normal. No axis deviation.

Sinus tachycardia

In the electrocardiogram of sinus tachycardia (Fig. 14) the P wave conforms to the normal type, and it recurs at regular intervals unless sinus arrhythmia is present. It is followed by a ventricular complex which need not be abnormal. Similarly the P–R period is not necessarily altered (although it is often a little shortened) so that the stimulus commencing in the sinus node is conducted to the ventricles in the usual way. Thus, auriculoventricular systole is normal, but the diastolic period is shortened. The tracing is only abnormal in that the heart rate is rapid (over 100 a minute); it may be as rapid as 160.

Sinus arrhythmia

In the electrocardiogram of sinus arrhythmia (Fig. 15) the auricular and ventricular complexes conform to the normal type. The stimulus commences

in the sinus node and is conducted normally to the ventricle but it arises at irregular intervals because of variation in vagal activity Thus the frequency of the impulse is increased during the phase of inspiration from lessened vagal influence, and decreased during expiration when normal vagal influence is restored

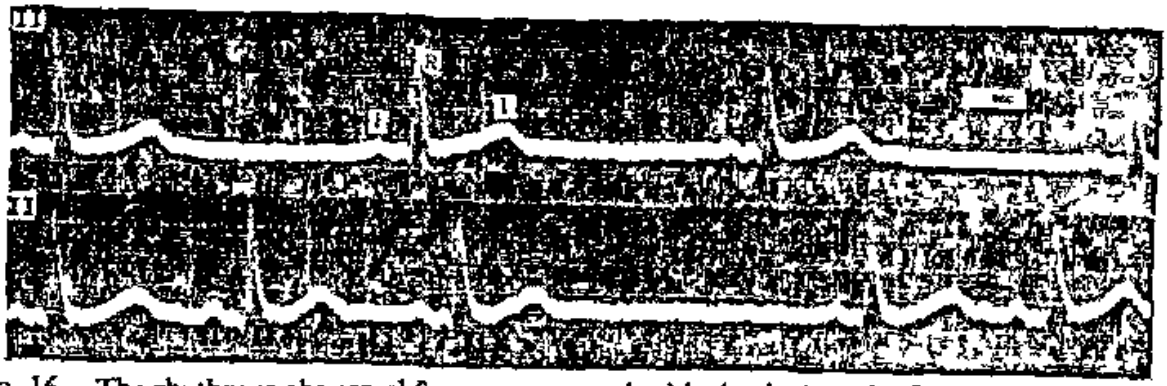


FIG 16 —The rhythm is abnormal from sino auricular block which in the first tracing has halved the heart rate

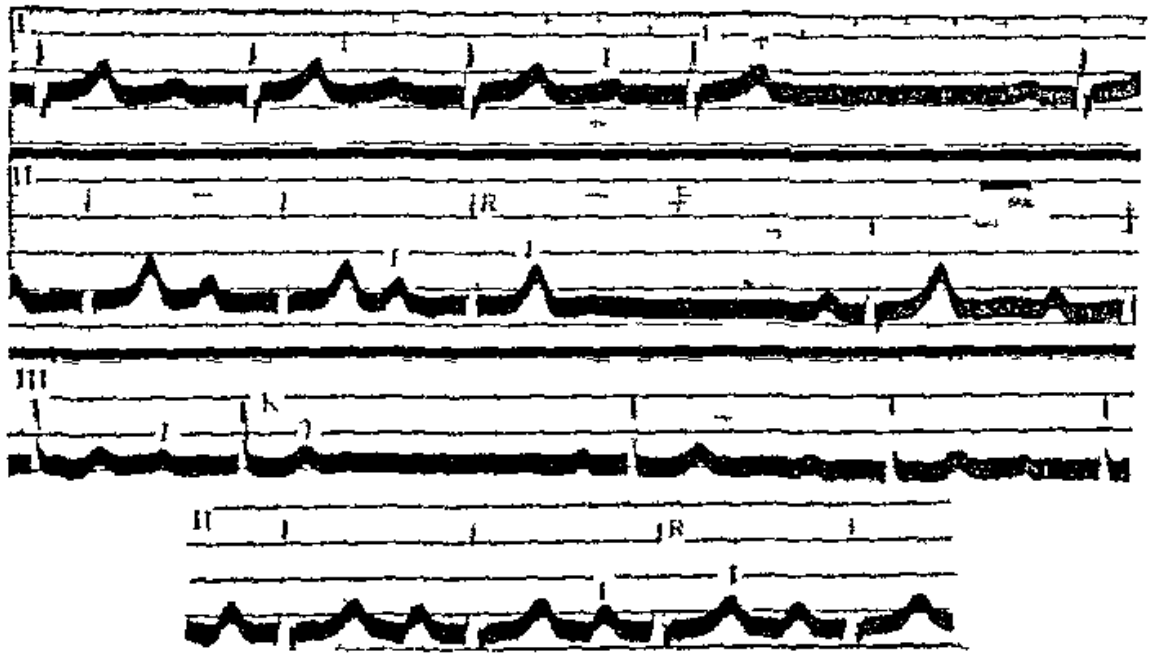


FIG 17 —The rate is normal The rhythm is irregular from sino-auricular block The P R period is very prolonged but this is lessened in the lower record following atropine No axis deviation The T wave is upright in all leads

Sino-auricular block

In the electrocardiogram of sino auricular block (Figs 16 and 17) a single beat fails to appear, although the silent period is generally a little shorter than two normal cycles The mechanism is probably due to suppression of the impulse originating in the sinus node by increased vagal action The condition differs from auriculoventricular block in that the auricular as well as the ventricular complex is missing

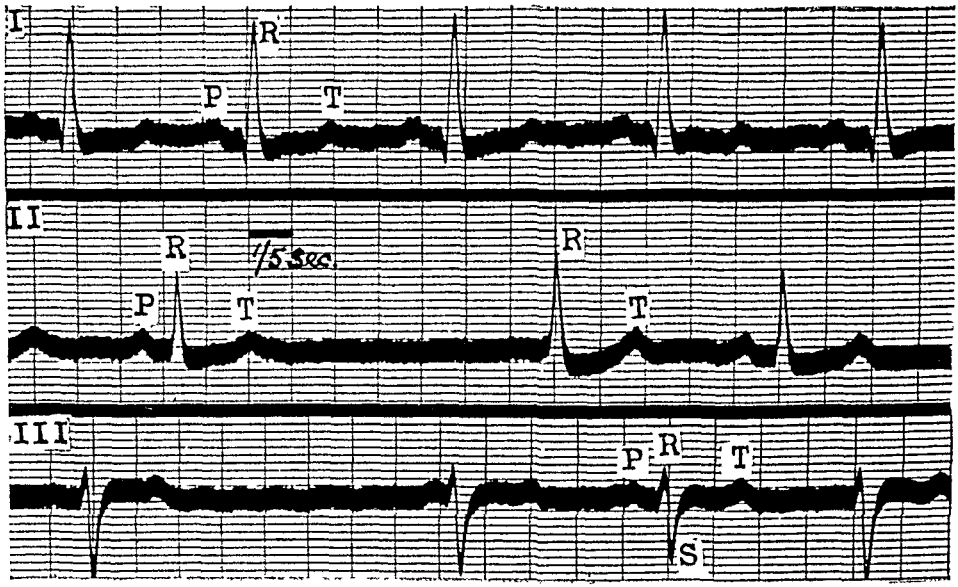


FIG. 18.—The rate is normal. The rhythm is irregular from ventricular escape in lead II. The P–R period is normal in other beats. L.A.D. The T wave is low in lead I.

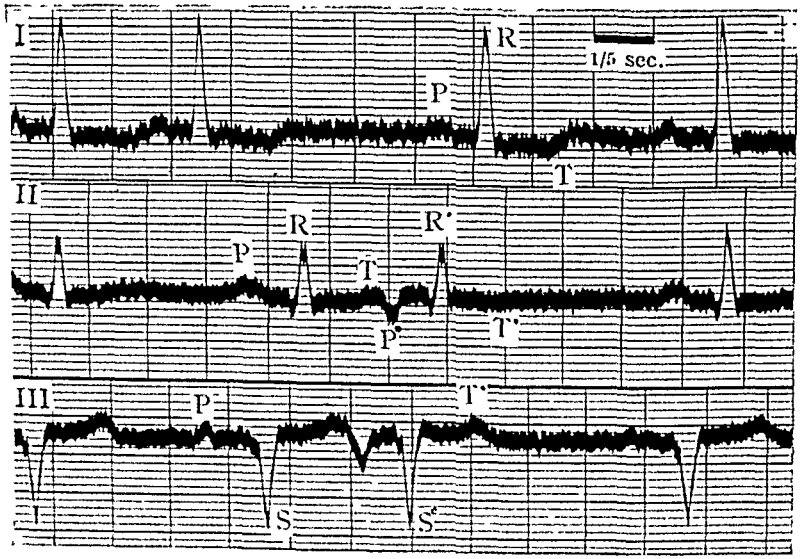


FIG. 19.—The rate is normal. The rhythm is irregular from auricular extrasystoles in which the premature P waves are inverted. The P–R period is normal. L.A.D. The T wave is inverted in lead I and low in lead II.

Ventricular escape

In the electrocardiogram depicting this condition (Fig 18) a P wave is missing and the ventricular complex is delayed and appears alone Sometimes the tracing may also show sinus bradycardia Absence of auricular activity may apply to a single beat, but rarely it may characterize a group of beats Should it be the dominant rhythm, the rare condition of auricular standstill is established

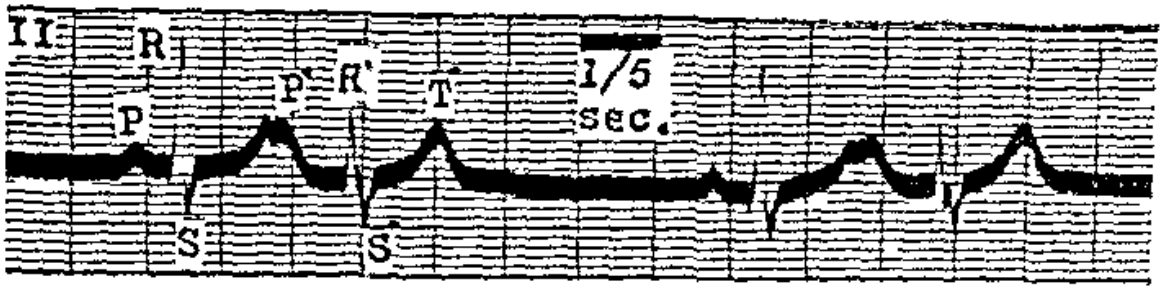


FIG 20 —The rhythm is irregular from alternating auricular extrasystoles where the premature P waves are formed at the end of the T waves

SHIFTING OF FOCUS NORMALLY INITIATING CARDIAC IMPULSE

Extrasystoles

The ectopic stimulus giving rise to extrasystoles may start in the auricle auriculo ventricular node, or ventricle The impulse commences prematurely and the contraction which it initiates suppresses the next normal impulse in the sinus node so that the extrasystole is followed by a pause before the succeeding normal

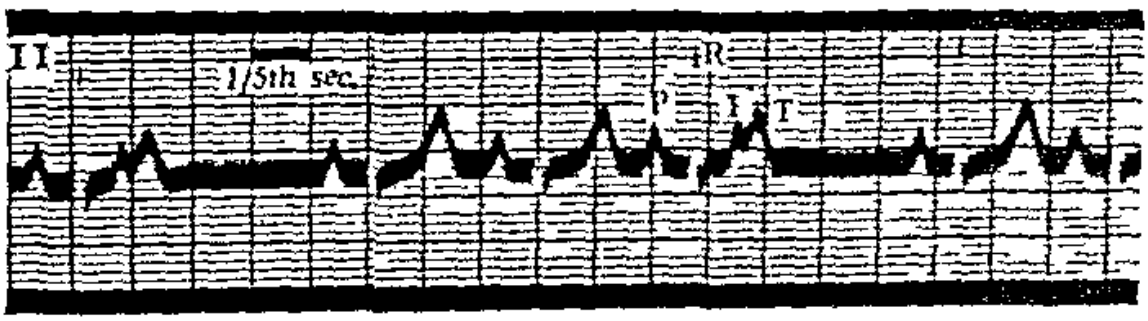


FIG 21 —The rhythm is irregular from blocked auricular extrasystoles The premature P waves, placed between the R and T waves, are not followed by the customary ventricular complex

beat takes place Rarely, and usually when the heart rate is slow a premature beat may be completed before the normal stimulus in the sinus node produces auricular systole, so that no pause follows in this instance the extrasystole is an added beat and is said to be *interpolated* between two normal beats

In auricular extrasystole (Figs 19 and 20) the P wave occurs prematurely and since it results from a stimulus arising in part of the auricle removed from the sinus node, it is as a rule deformed and often inverted The P–R period is usually shortened and the ventricular complex shows a slight variation from the normal,

The pause which follows this and other kinds of extrasystoles is longer than the usual diastolic period and is referred to as the compensatory pause.

When the auricular beat is so premature that the impulse arrives in the ventricle before it has relaxed from the preceding systole, the ventricle does not respond to the stimulus; this condition is known as blocked auricular extrasystole (Fig. 21).

In **auriculoventricular nodal extrasystole** (Fig. 22) the P wave, which is usually deformed, is placed after or within the ventricular complex which may also show a slight variation from the normal. The chest lead CR_1 often depicts the obscured P wave more clearly than do other leads (Fig. 23).

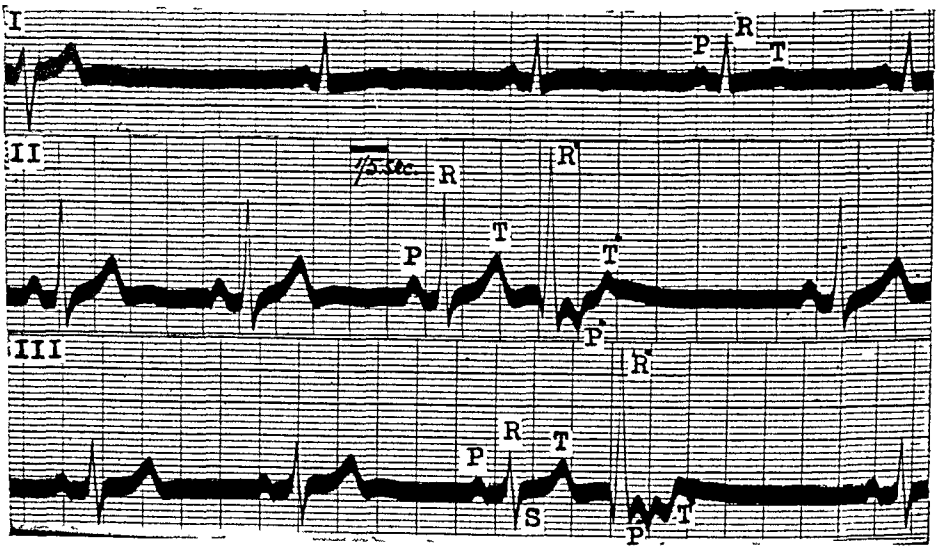


FIG. 22.—The rate is normal. The rhythm is irregular from nodal extrasystoles in which the P wave is written between the R and T waves. In other beats the P–R period is normal. No axis deviation. The T wave is low in lead I.

In **ventricular extrasystole** the customary rhythm is interrupted by a complex which is premature and which is not preceded by a P wave. It consists of a wide QRS complex continued into a T wave which is generally written in the opposite direction to the QRS deflection. The P wave is included either in the wide QRS or in the exaggerated secondary T wave. In the case of an extrasystole arising in the right ventricle (Fig. 24) the abnormal QRS complex is relatively high in lead I, deep in lead III, and diphasic and often of low voltage in lead II. In the case of an extrasystole arising in the left ventricle (Fig. 25) the abnormal QRS complex is tall in leads II and III and low or directed downwards in lead I.

Ventricular premature beats, however, often fail to conform to such characteristic types and it may be that these take origin in the septum or junctional tissues.

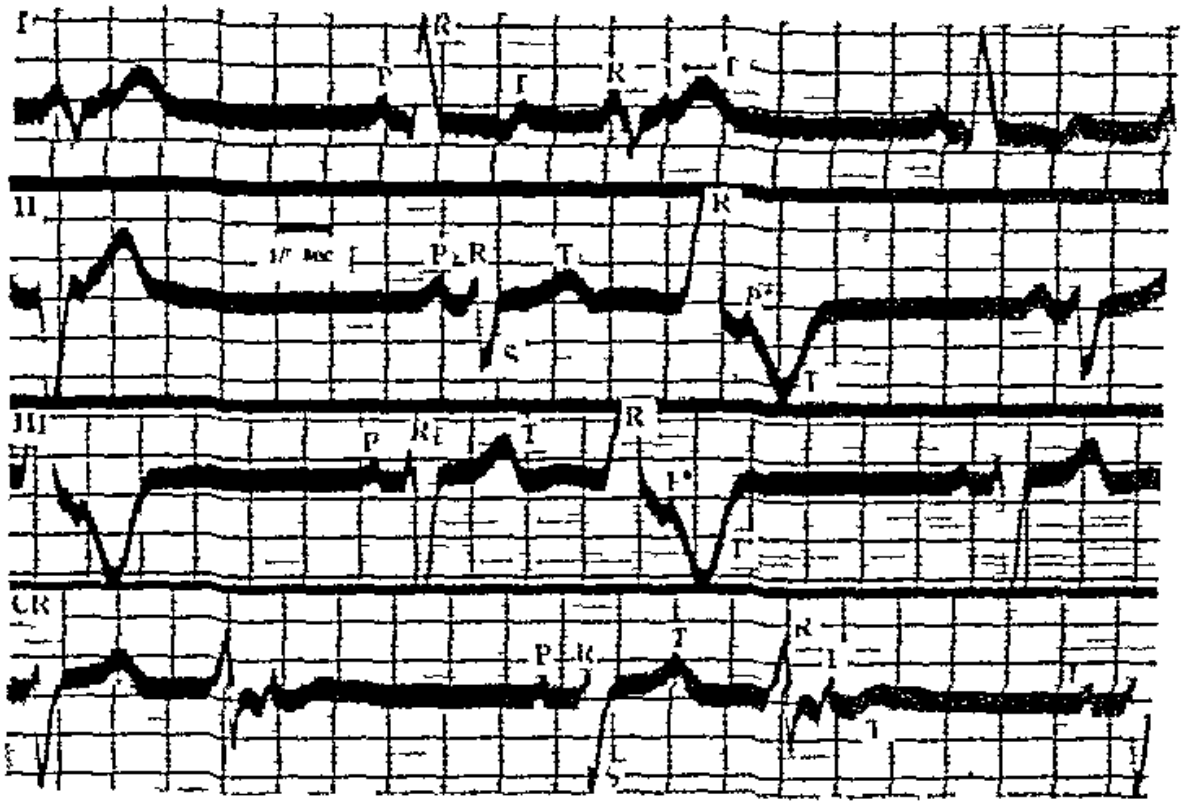


FIG 23.—The rate is not rapid. The rhythm is abnormal from alternating nodal extrasystoles where the P wave following the QRS is best depicted in CR. The P R period is not abnormal. L.A.D. The T wave is inverted in lead I with slight R T depression from hypertension.

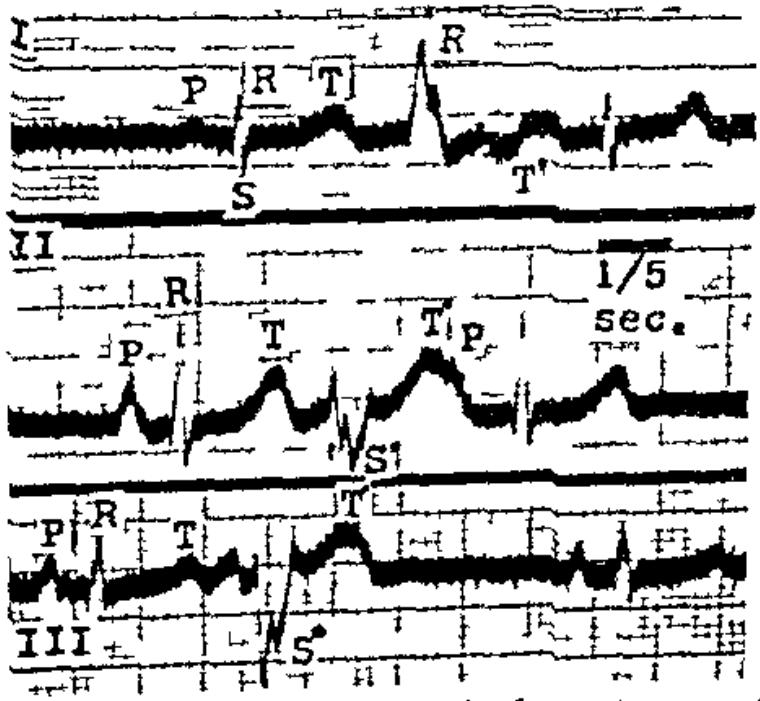


FIG. 24.—The rate is normal. The rhythm is irregular from right ventricular extrasystoles. The P R period is normal. No axis deviation. The P wave is tall and spiky in lead II.

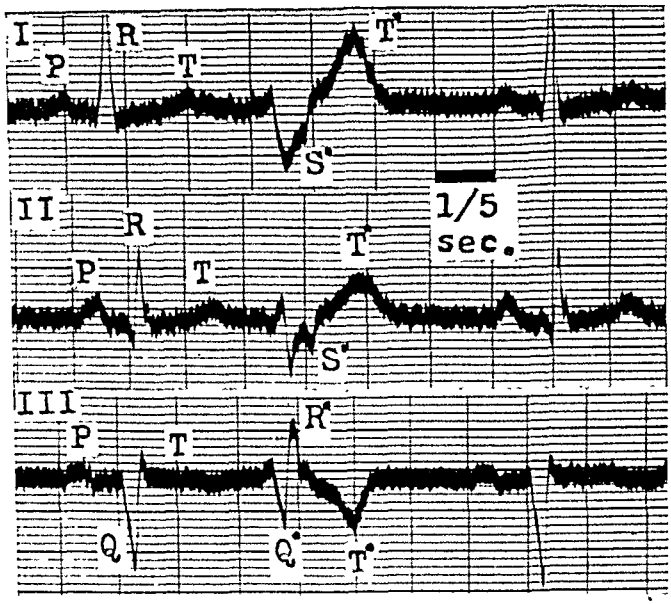


FIG. 25.—The rate is normal. The rhythm is irregular from left ventricular extrasystoles. The P-R period is normal. L.A.D. The Q wave is deep and the T wave low in lead III.

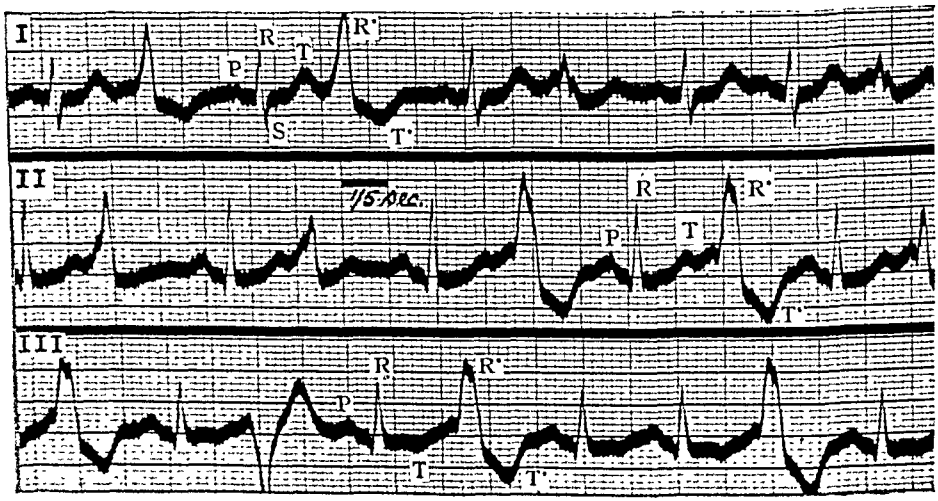


FIG. 26.—The rate is a little rapid. The rhythm is irregular from frequent extrasystoles. The P-R period is normal. No axis deviation. The T wave is inverted in lead III.

As a rule extrasystoles are abolished by induced tachycardia but sometimes (Fig 26) the two varieties of arrhythmia exist side by side

When the electrocardiogram is equivocal in cardiac infarction as for instance in bundle branch block the form of an extrasystole may provide a clue to such a diagnosis (Fig 27)

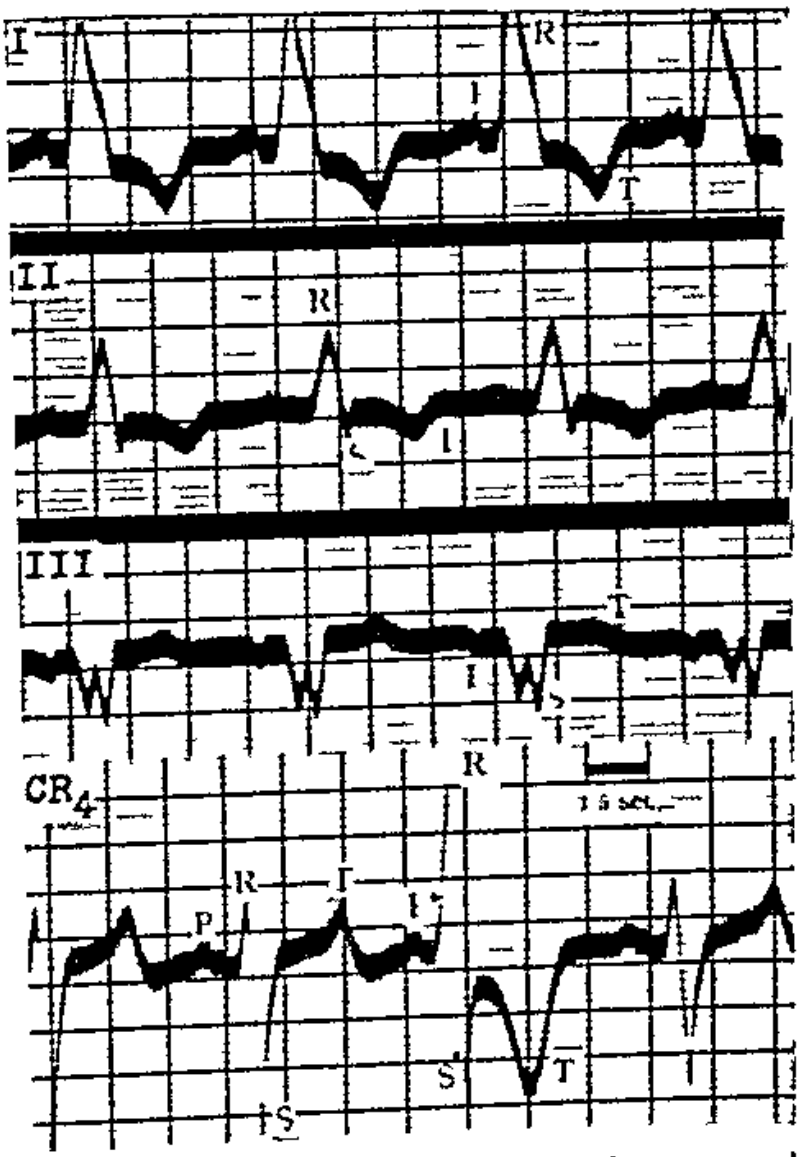


FIG 27.—The rate is normal The rhythm is normal except for one auricular extrasystole in CR_4 The P R period is normal L A D The QRS complexes are wide and the T waves are written in the opposite direction of the QRS The form of the T wave in the extrasystole suggests cardiac infarction (anterior)

Auriculoventricular nodal rhythm

In the electrocardiogram of A V nodal rhythm the P wave usually follows the QRS complex although it often coincides with or precedes the R wave Since the impulse commences in the A–V node and passes backwards into the auricle as well as forwards into the ventricle the P wave may be deformed The form

of the QRS complex is often modified in many beats by the proximity of the auricular contraction. Usually the rate is either normal or slower (Fig. 28), but sometimes it is more rapid (Fig. 29).

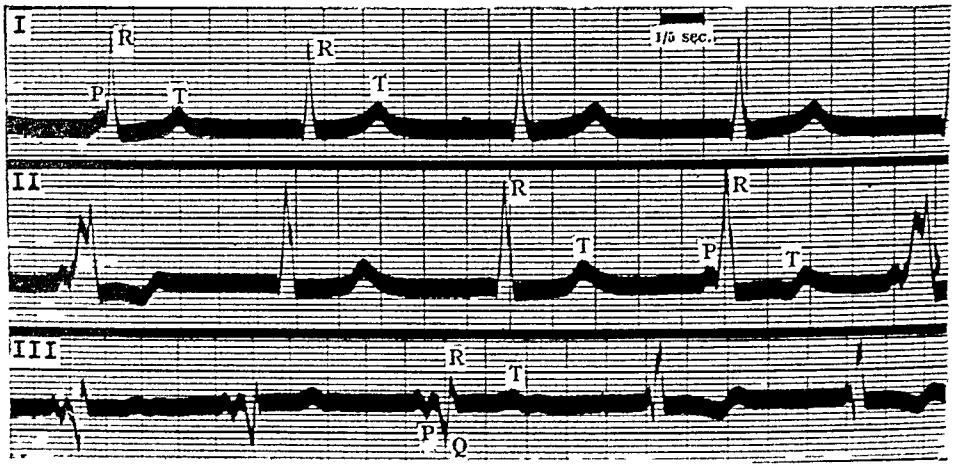


FIG. 28.—The rate is a little slow. The rhythm is irregular from nodal rhythm where the focus initiating the cardiac impulse is variable (shifting pace-maker). The QRS complexes widen when they are preceded by the P wave. The T wave for the same reason is sometimes inverted. No axis deviation.

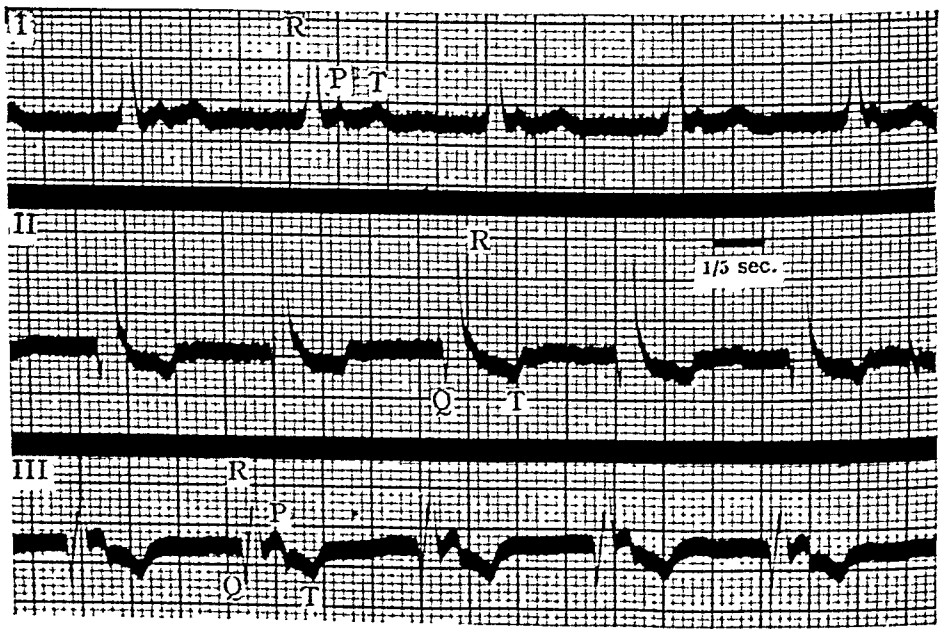


FIG. 29.—The rate is a little rapid. The rhythm is abnormal from nodal rhythm where the P wave follows the QRS complex. No axis deviation. Prominent Q waves and inverted T waves in leads II and III from cardiac infarction.

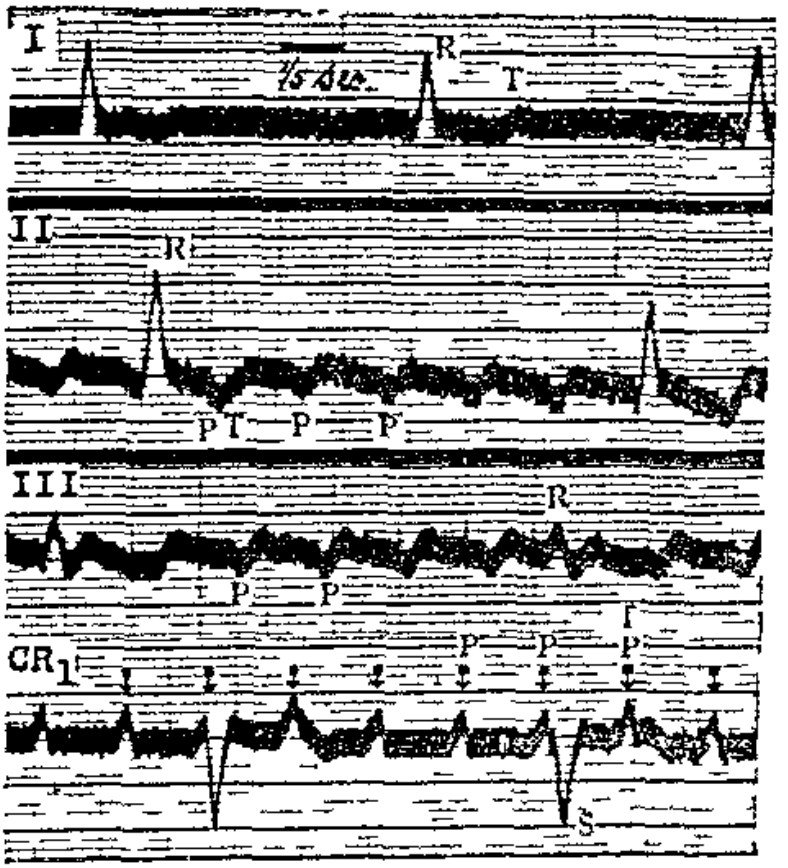


FIG 30 —The ventricular rate is slow The rhythm is irregular from auricular tachycardia of high rate with 6 to 1 A-V dissociation No axis deviation The R-T segments are depressed by digitalisation

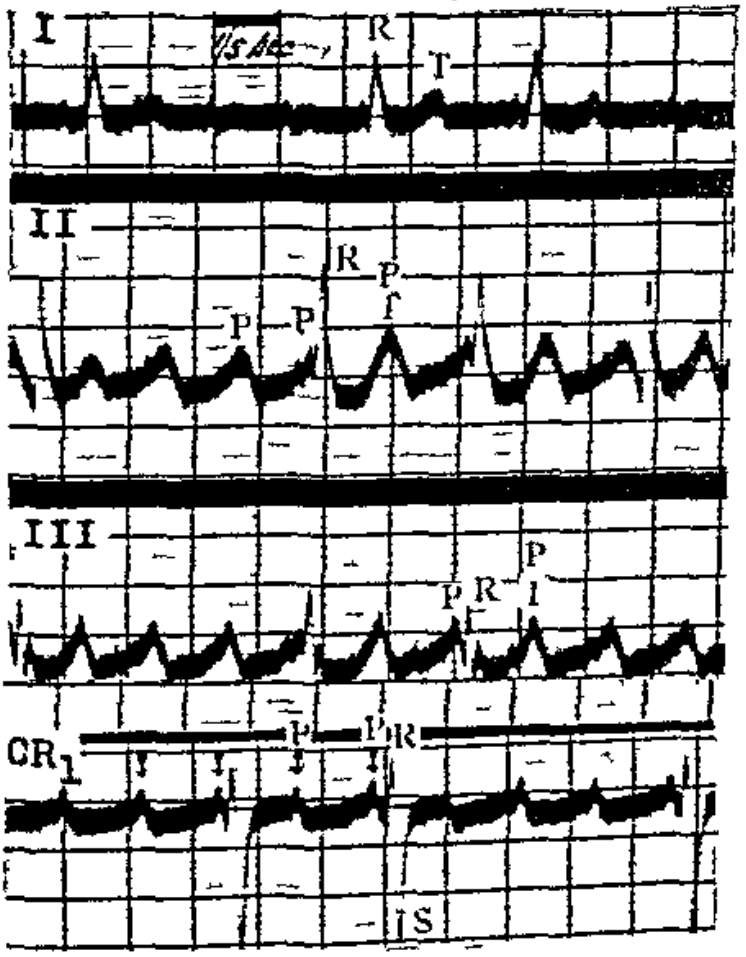


FIG 31 —The ventricular rate is a little rapid The rhythm is irregular from auricular tachycardia of high rate with 2 or 3 to 1 A-V dissociation No axis deviation

Paroxysmal tachycardia and auricular flutter

The auricular rate in paroxysmal or auricular tachycardia varies from 250 to 500 a minute, but when the ventricular rate is also rapid, all the auricular contractions are difficult to identify ; the recognition of the auricular beats, however, is facilitated by recording the right pectoral chest lead (CR_1) during tachycardia and comparing it with the record obtained in normal rhythm. When this test becomes routine a high incidence of A–V dissociation will be found among the

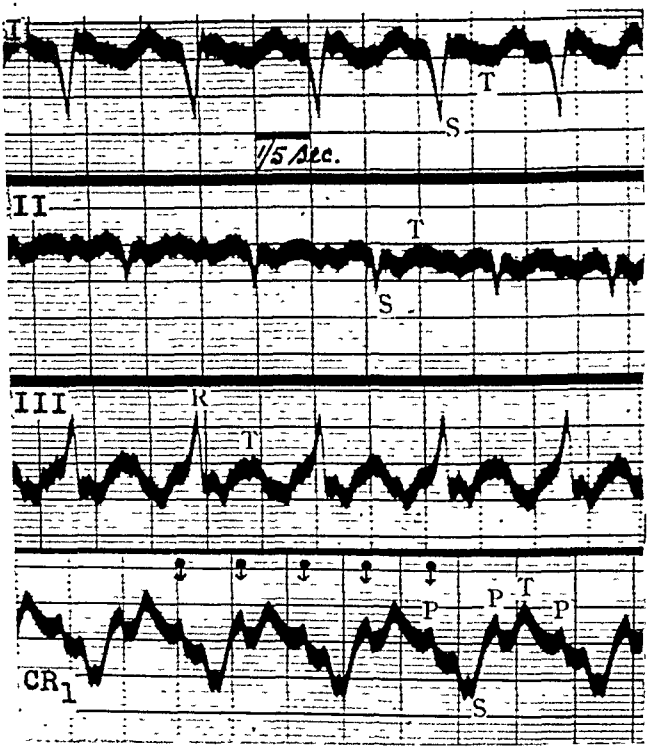


FIG. 32.—The rate is rapid. The rhythm is irregular from auricular tachycardia of high rate with 2 to 1 A–V dissociation which is best seen in CR_1. R.A.D. The T wave is depressed in lead I.

cases. A regard of the auricular rate and the degree of A–V dissociation, both of which determine the conspicuity of the auricular waves in the tracing, places the electrocardiograms of auricular tachycardia in five groups. In the past it has been traditional to allude to the first two groups as auricular flutter, but the mechanism is the same for these as for the next two groups.

1. **Auricular tachycardia of high rate with 3 to 1 (or greater) A–V dissociation.**
   In this group the auricular rate is rapid (up to 300 a minute), but the ventricular rate is relatively slow (under 100) so that A–V dissociation is 3 to 1 or greater, and three or more consecutive auricular waves are written outside the ventricular waves (Figs. 30 and 31).

2. **Auricular tachycardia of high rate with 2 to 1 A–V dissociation.**
   In this group the auricular rate varies from 200 to 260, and the ventricular rate from 100 to 130 ; both auricular waves are seen outside the ventricular waves (Figs. 32 and 33).

3 *Auricular tachycardia of higher rate with 2 to 1 A V dissociation*

In this group the auricular rate varies from 260 to 400 and the ventricular rate from 130 to 200 alternate auricular waves fall within the S or T waves of the ventricular complex (Fig 34)

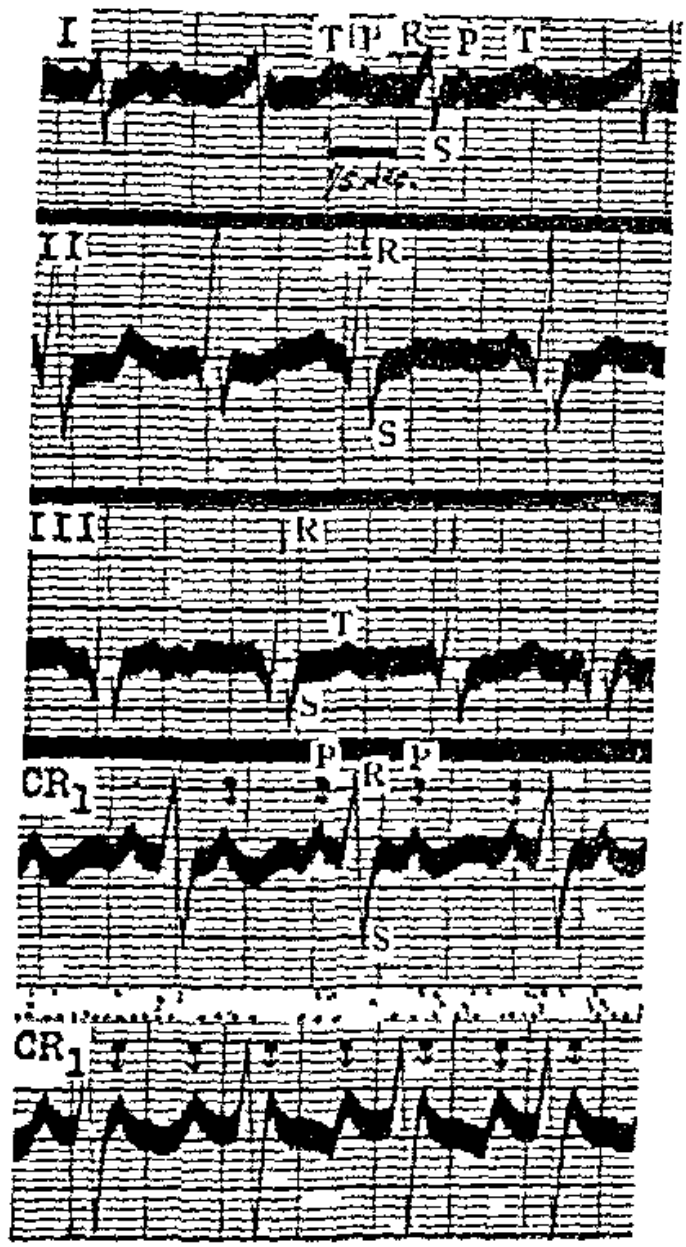


FIG 33.—The rate is rapid The rhythm is irregular from auricular tachycardia of high rate with 2 to 1 A V dissociation which is best seen in CR_1; two such leads are shown illustrating how a slower ventricular rate permits a better view of the two auricular waves

4 *Auricular tachycardia of very high rate with 2 to 1 A V dissociation*

In this group the auricular rate is over 400 and the ventricular rate over 200 both auricular waves fall within the S and T waves of the ventricular complex (Fig 35)

5 Auricular tachycardia of high rate without A V dissociation

In this group the auricular rate is rapid and the ventricular rate is the same so that the impulse from the ectopic focus travels through the auricular node (*paroxysmal tachycardia*) Such paroxysms are brief as a rule and recur frequently (Fig 36) Regular responses of ventricular contraction to each auricular contraction may also be obtained by adrenaline in a patient exhibiting auricular tachycardia with A V dissociation (Fig 37)

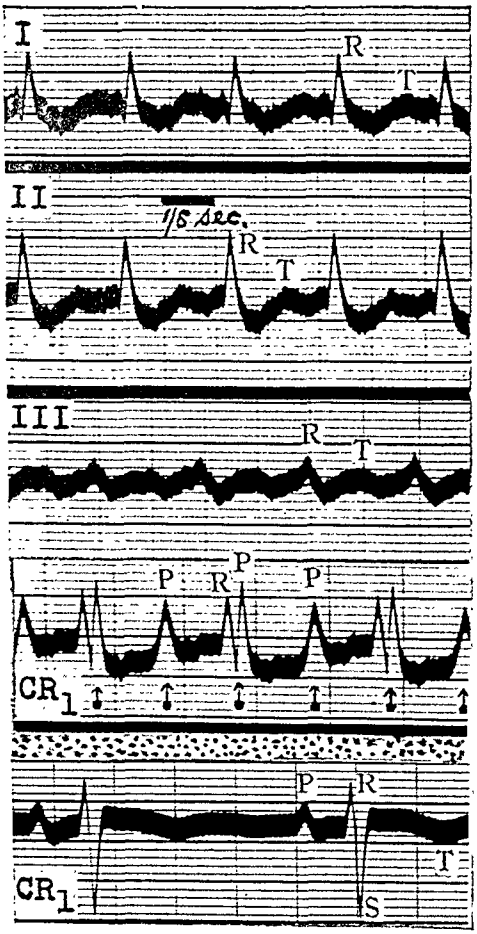


FIG. 34.—The rate is rapid. The rhythm is irregular from auricular tachycardia of high rate with 2 to 1 A–V dissociation which is only seen in CR_1. No axis deviation. The T wave and R–T segments are depressed and the T wave remains inverted in the lower CR_1 which was recorded after the attack.

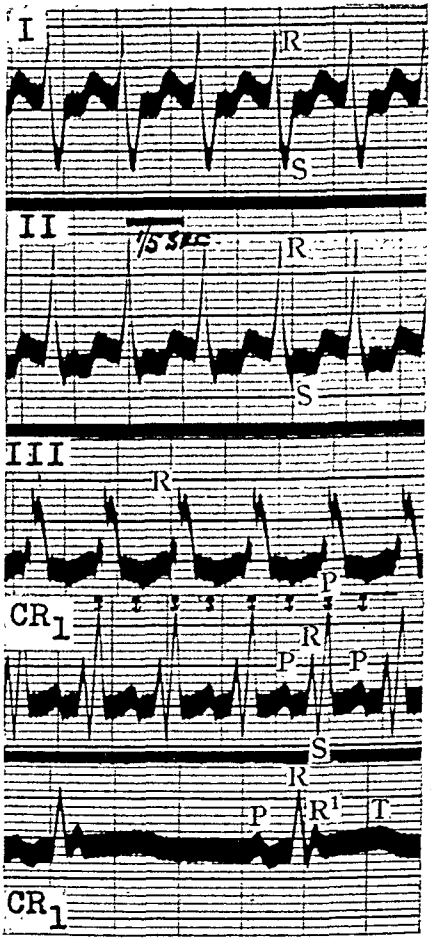


FIG. 35.—The rate is rapid. The rhythm is irregular from auricular tachycardia of very high rate with 2 to 1 A–V dissociation which is only seen in CR_1. Tendency to R.A.D. The R–T segments are depressed. The lower CR_1 is in normal rhythm.

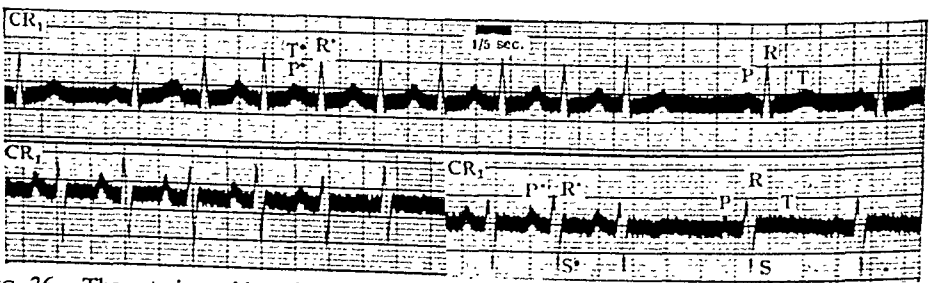


FIG. 36.—The rate is rapid at times. The rhythm is sometimes normal and sometimes irregular from auricular tachycardia of high rate without A–V dissociation, that is, parasinus tachycardia.

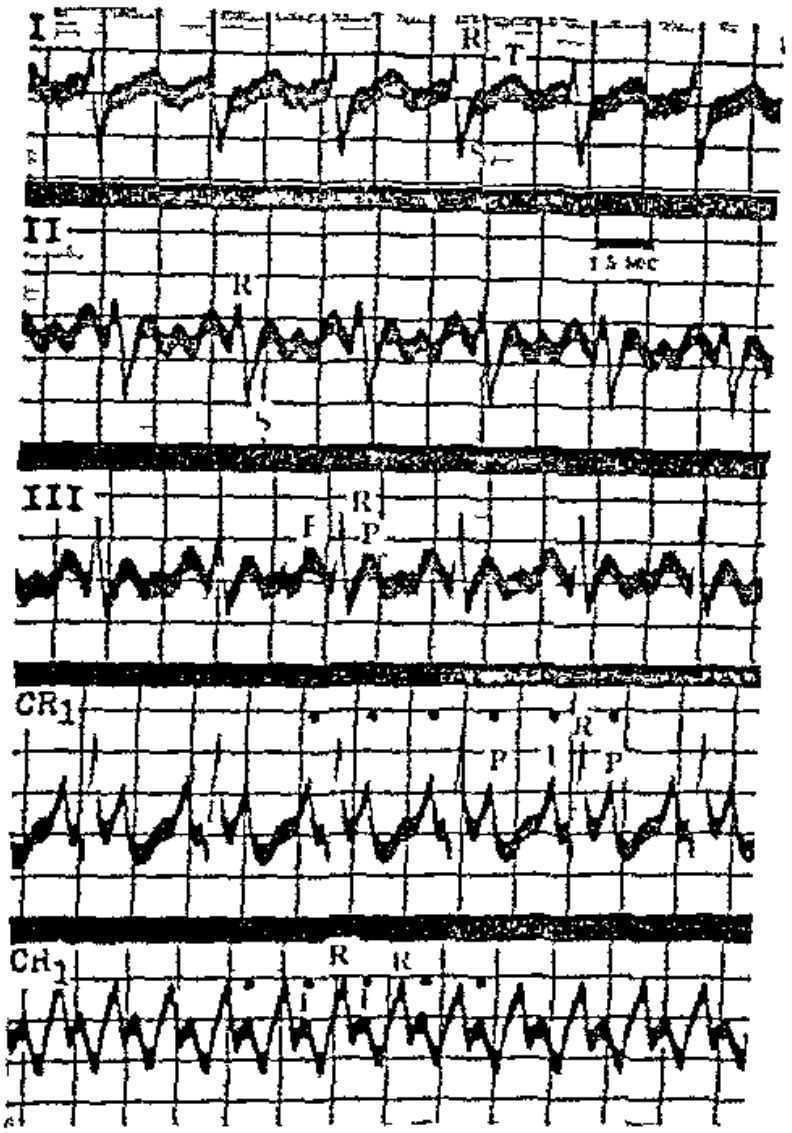
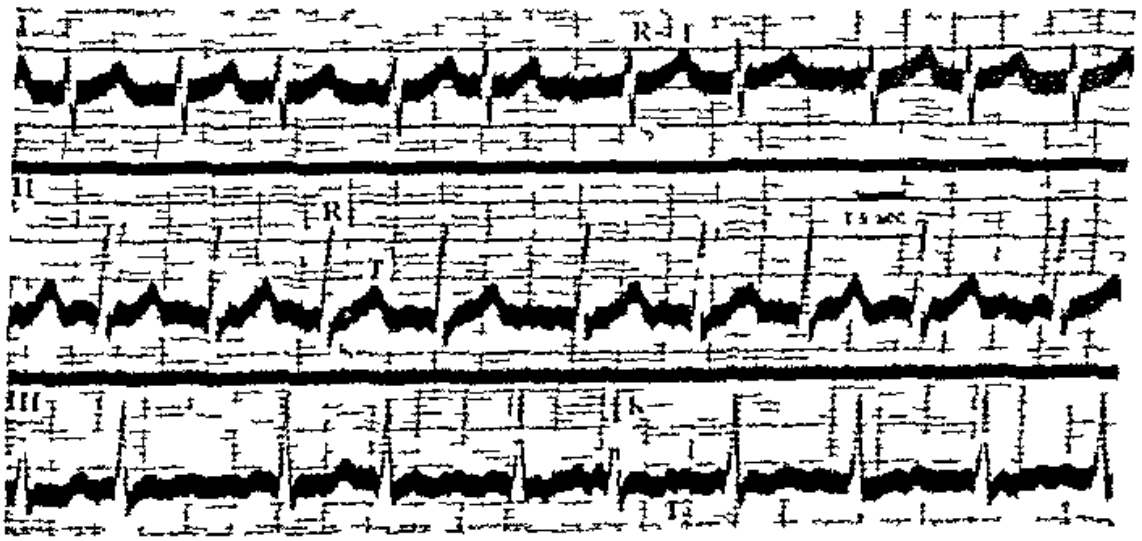


FIG 37.—*The rate is rapid* The rhythm is irregular from auricular tachycardia of a high rate with 2 to 1 A-V dissociation which shows in lead III but is more obvious in CR_1 In the lower CR_1 the ventricular rate has been doubled to assume the same rate as the auricle after an injection of adrenaline

FIG 38.—The rate is rapid *The rhythm is irregular* from auricular fibrillation where the f waves are small R A D The T wave is upright in all leads



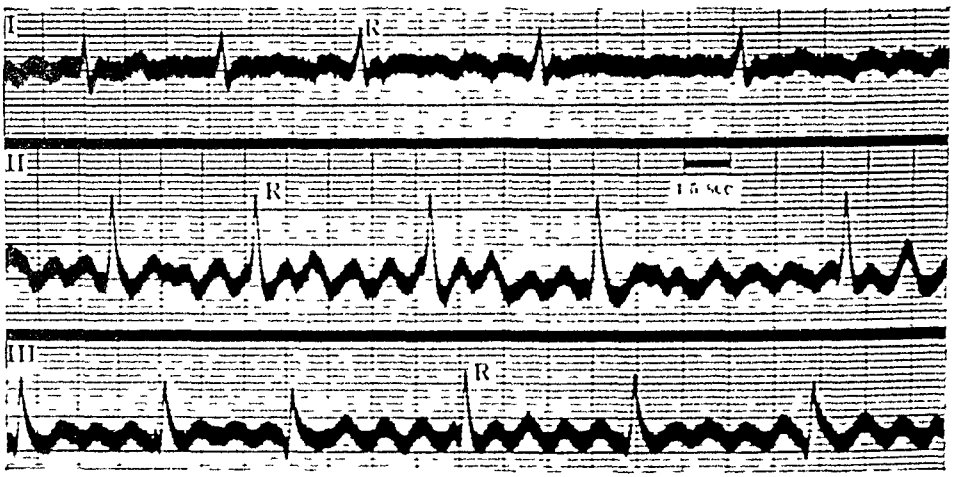


FIG. 39.—The rate is not rapid. The rhythm is irregular from auricular fibrillation where the "f" waves are prominent. No axis deviation. The T wave is low in lead I.

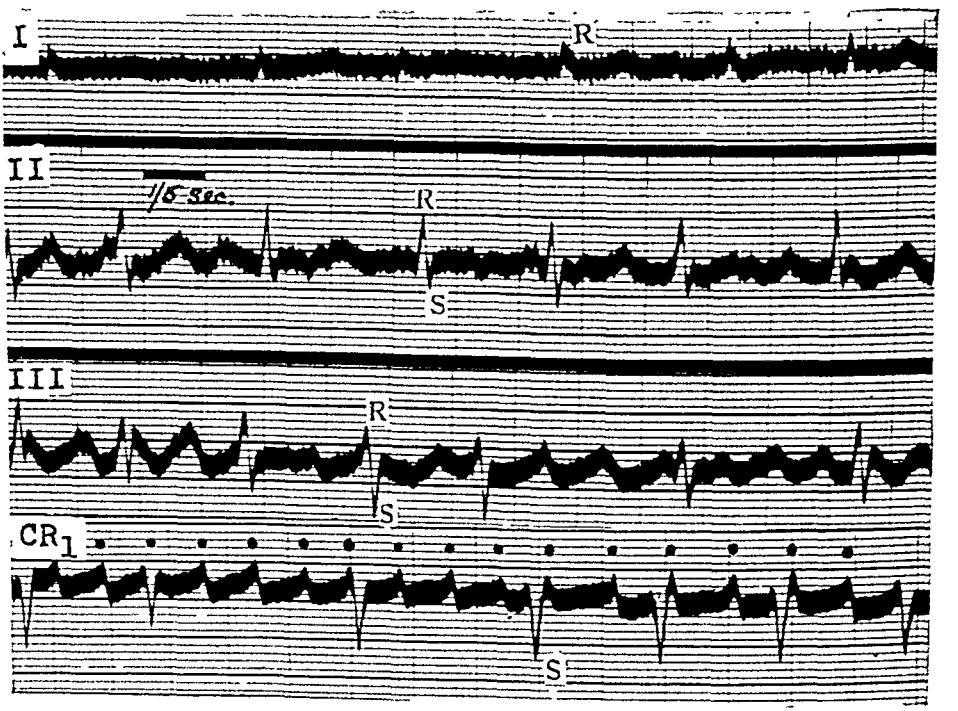


FIG. 40.—The rate is a little rapid. The rhythm is irregular from auricular fibrillation. The "f" waves are prominent in CR_1. No axis deviation. Small voltage in lead I. The R–T segments are depressed by digitalis.

Auricular fibrillation

In the limb lead electrocardiogram of auricular fibrillation the P waves are replaced by oscillations which may be hardly visible (Fig 38) or fairly prominent

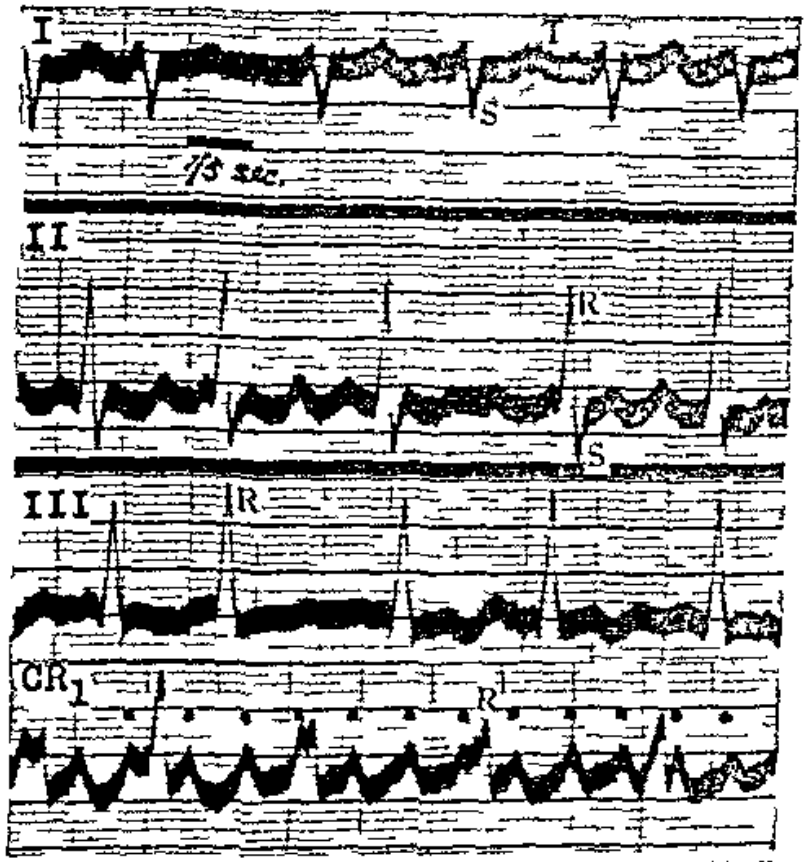


FIG 41.—The rate is a little rapid. The rhythm is irregular from auricular fibrillation. The T waves are prominent and for the most part recur regularly in CR_1. The R T segments are depressed by digitalis

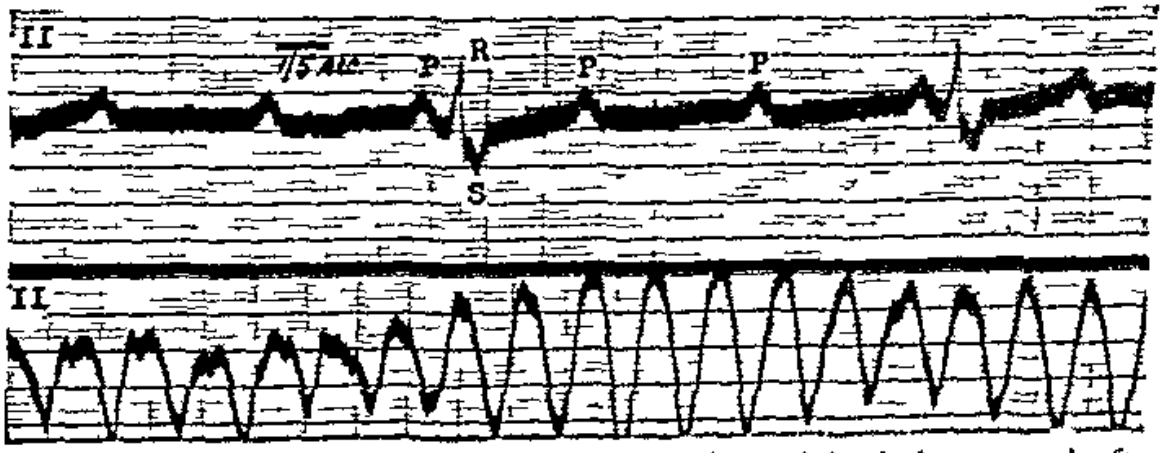


FIG 42.—In the first tracing the ventricular rate is very slow and the rhythm is irregular from complete heart block. In the second tracing the rate is very rapid and the complexes are gross and irregular from ventricular fibrillation

(Fig 39) The auricular beats are seen to the best advantage in the right pectoral electrocardiogram (CR_1) where they may appear as well formed waves differing slightly in size and shape and with a rhythm almost as often regular as irregular

(Figs. 40 and 41). The ventricular responses in fibrillation have no constant relationship to the auricular waves.

Ventricular fibrillation

The electrocardiogram of ventricular fibrillation (Fig. 42) is characterized by frequent, large and irregular oscillations. Amongst these it is not possible to identify any auricular waves. Apart from the short period preceding death from such illness as cardiac infarction, ventricular fibrillation is characteristically seen in relation to the Stokes-Adams attack in complete heart block.

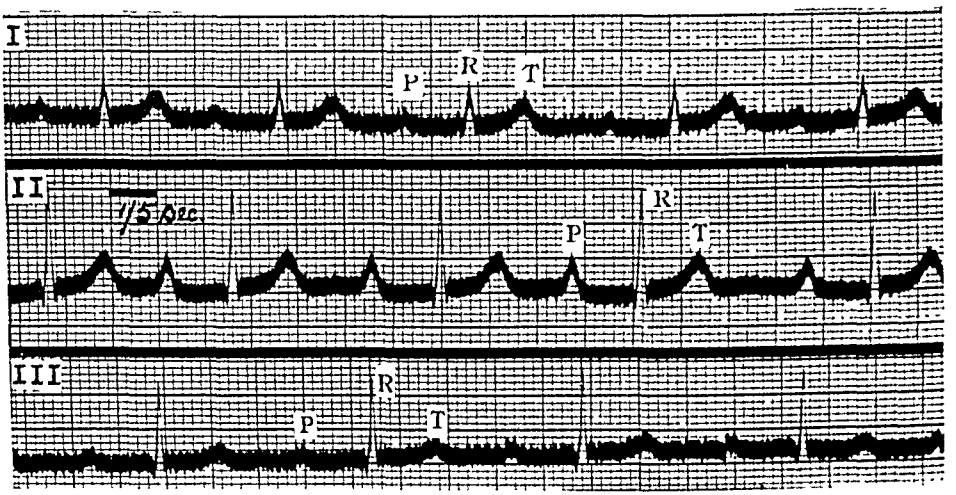


FIG. 43.—The rate is normal. The rhythm is regular. The P–R period is prolonged. No axis deviation. The T wave is low in lead III.

FAULTY CONDUCTION OF THE CARDIAC IMPULSE

Prolonged P-R period

The time taken for the impulse to travel from the sinus node in the auricle to the ventricle is represented by the length of the P–R period measured from the commencement of the P wave where it leaves the isoelectric level to the commencement of the R wave. The normal P–R period varies between 0·1 and 0·2 second or at the most 0·22 second. When the P–R period is prolonged the electrocardiogram may be normal in other respects (Fig. 43), while this delayed A–V conduction is common in bundle branch block (Fig. 44). If the P–R period is exceptionally long or diastole is short, the P and T waves may coincide (Fig. 45); in this event the right pectoral lead is the best to portray both waves. Variation in the length of diastole from sinus arrhythmia will also help to diagnose the condition (Fig. 46).

Short P-R period

The P–R period is often short in nodal rhythm, but in this case its length may vary from beat to beat.

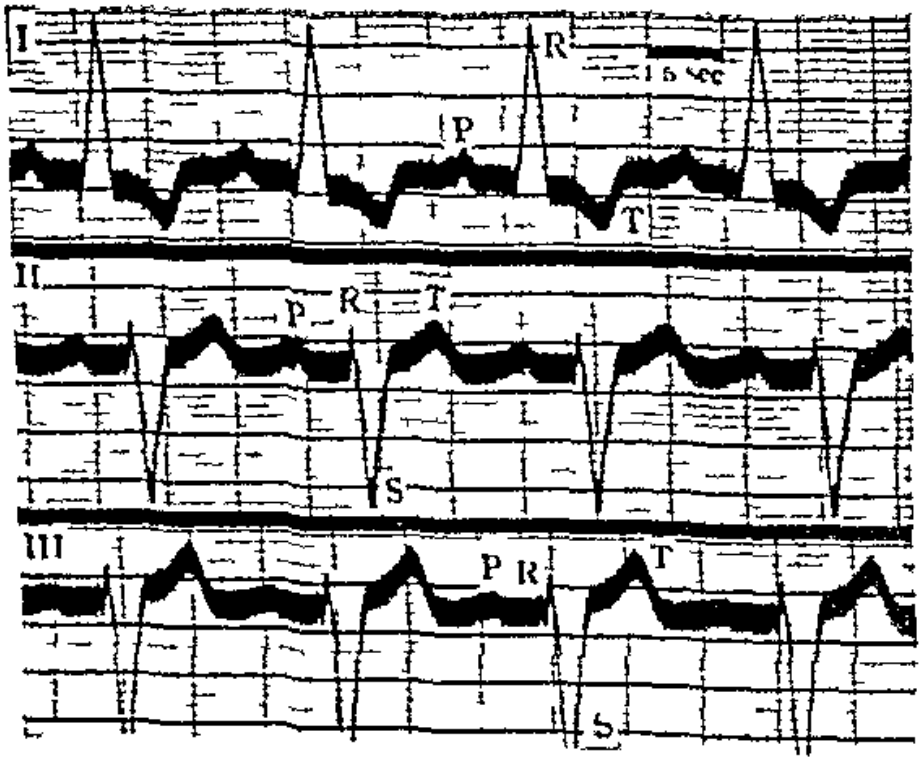


FIG 44.—The rate is a little rapid. The rhythm is regular. The P-R period is prolonged. LAD and the QRS is wide with the T wave written in the opposite direction (left bundle branch block).

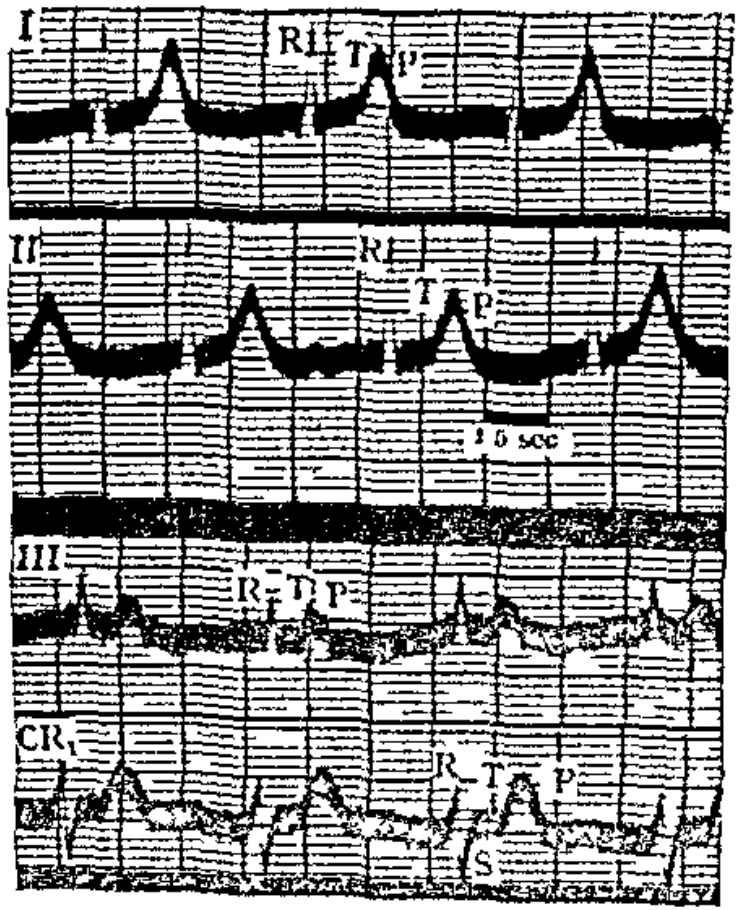


FIG. 45.—The rate is normal. The rhythm is regular. The P-R period is prolonged and the P wave combines with the T wave; this effect is best recognized in leads III and CR. No axis deviation.

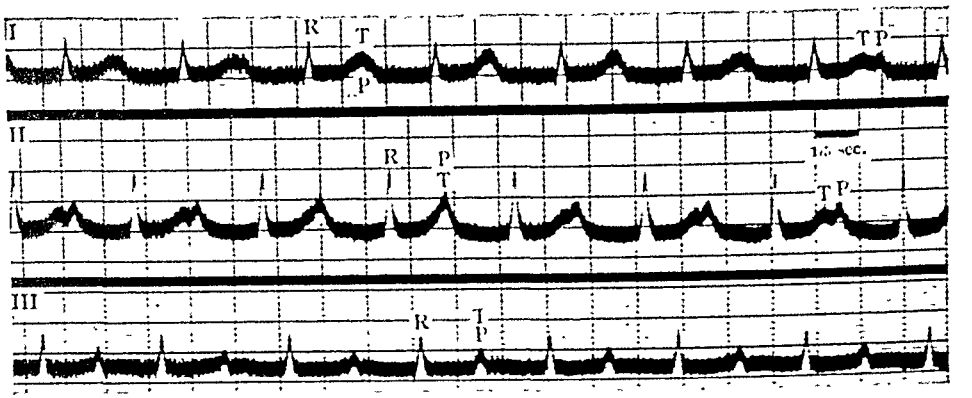


FIG. 46.—The rate is a little rapid. The rhythm is regular except for sinus arrhythmia. The P–R period is greatly prolonged and the P wave coincides with the T wave except when the diastolic period lengthens. No axis deviation. The T wave is upright in all leads. The R–T segments are normal.

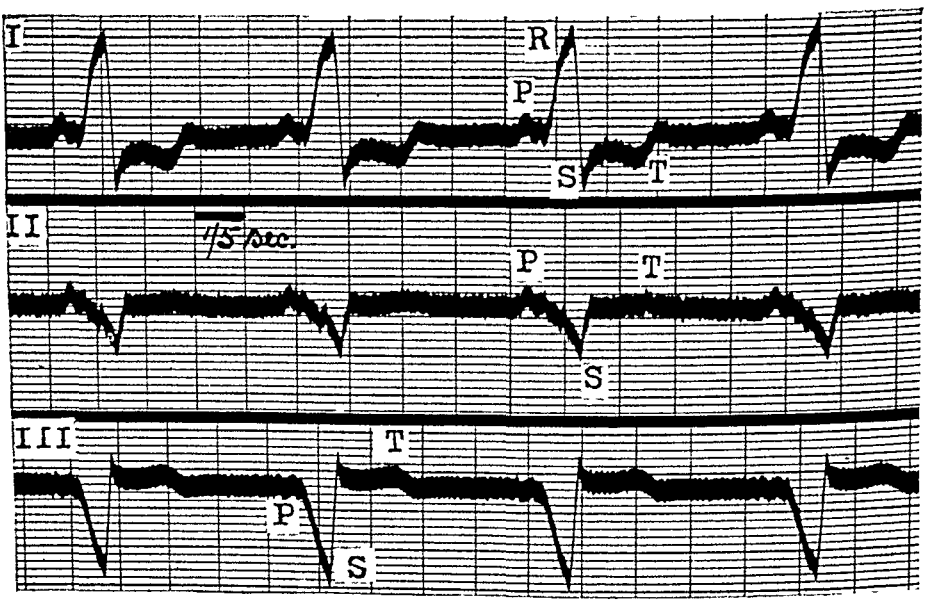


FIG. 47.—The rate is normal. The rhythm is regular. The P–R period is short. L.A.D. The QRS complexes are wide and the T wave is written in the opposite direction to the QRS.

When a short P R period is associated with bundle branch block (Fig 47) and paroxysmal tachycardia it constitutes what has become known as the Wolff Parkinson White syndrome and it may be found in otherwise healthy subjects

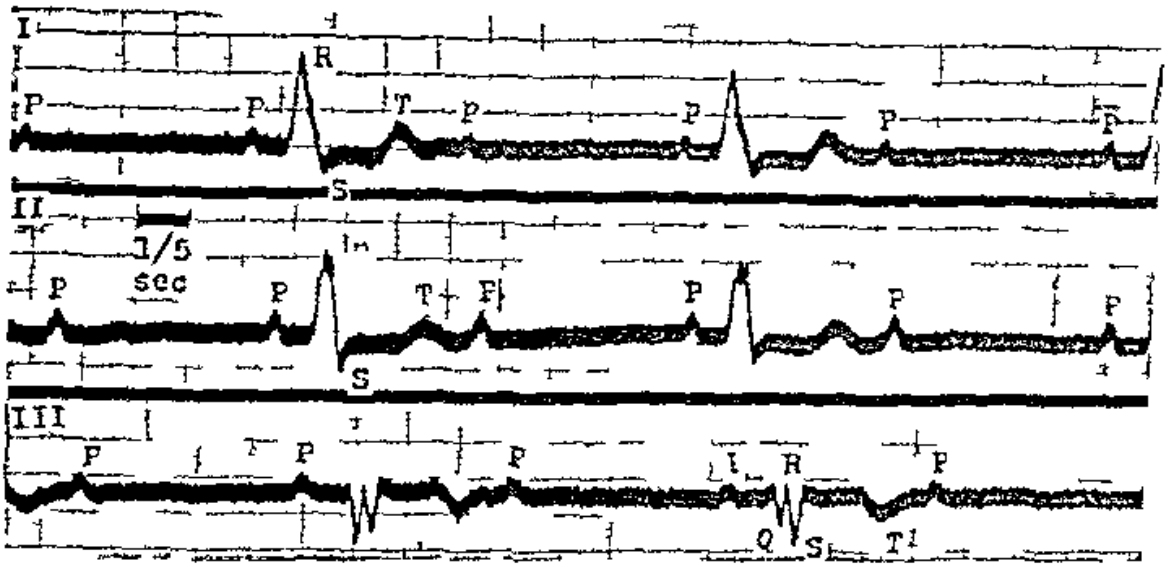


FIG 48.—The rate is slow. The rhythm is irregular from 2 to 1 A-V block. L.A.D. The QRS complexes are wide and the T wave is inverted in lead III.

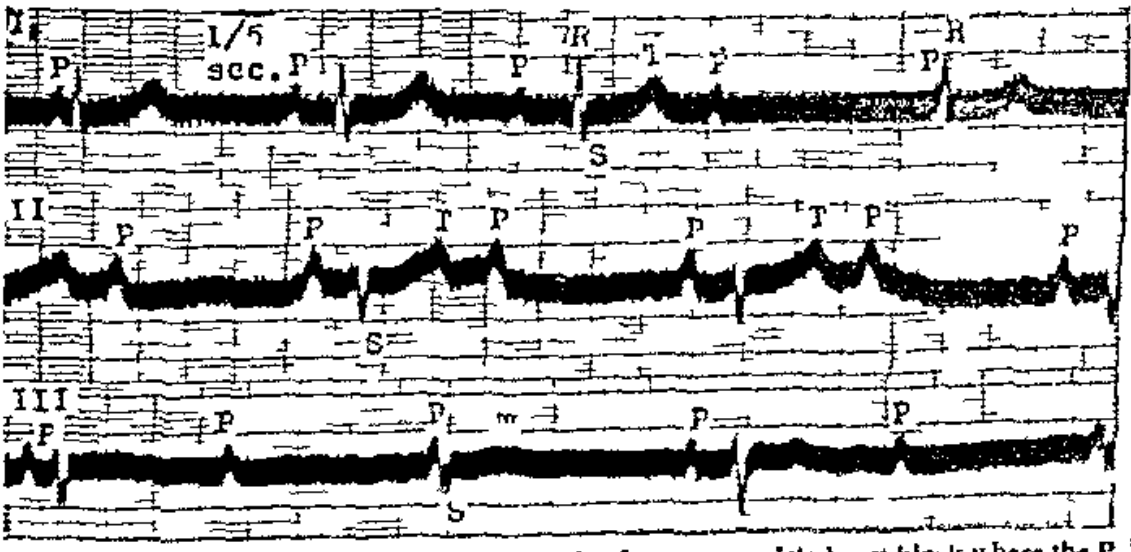


FIG 49.—The rate is slow. The rhythm is irregular from incomplete heart block where the P R periods in lead I lengthen in successive beats until a ventricular complex fails to appear (Wenckebach periods). No axis deviation.

Incomplete A-V block

The P wave and the QRS complex may be normal but each P wave is not followed by a ventricular wave because the ventricle fails to respond to a number of impulses arriving from the auricle. In the common variety the ventricle responds to alternate auricular beats (2 to 1 A-V block) (Fig 48). In the uncommon variety

the P–R lengthens progressively until finally an expected ventricular beat fails to appear (Fig. 49); after the pause the P–R period is shorter but it again lengthens in successive beats; usually the increase of the second over the first interval is greater than the increase of the third over the second, and the ratio between

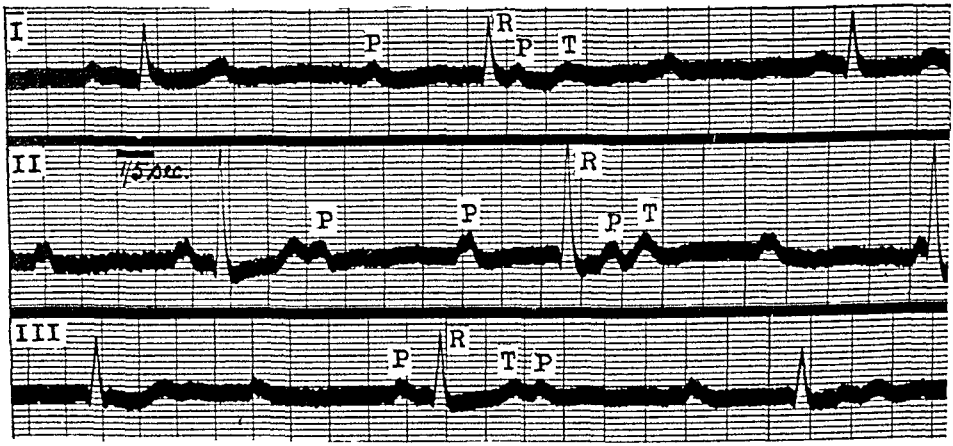


FIG. 50.—The rate is very slow. The rhythm is irregular from complete heart block. No axis deviation. The P waves are broad and occasionally bifid.

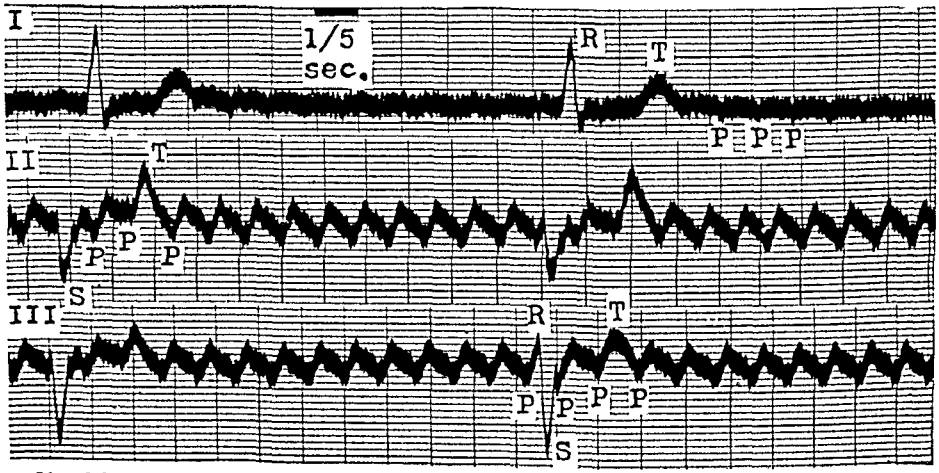


FIG. 51.—The ventricular rate is very slow, and the auricular rate is rapid from auricular tachycardia. L.A.D.

auricular and ventricular beats is 3 to 2 or 4 to 3. This variety is sometimes referred to as the Wenckebach phenomenon.

Complete A-V block

The electrocardiogram (Fig. 50) shows complete dissociation between the auricular and ventricular waves. Both auricular and ventricular beats recur

regularly though independently, and the auricular rate is more rapid (about 70 a minute) than the ventricular rate (30 or under) The QRS and T waves frequently conform to those found in bundle branch block The P wave is written

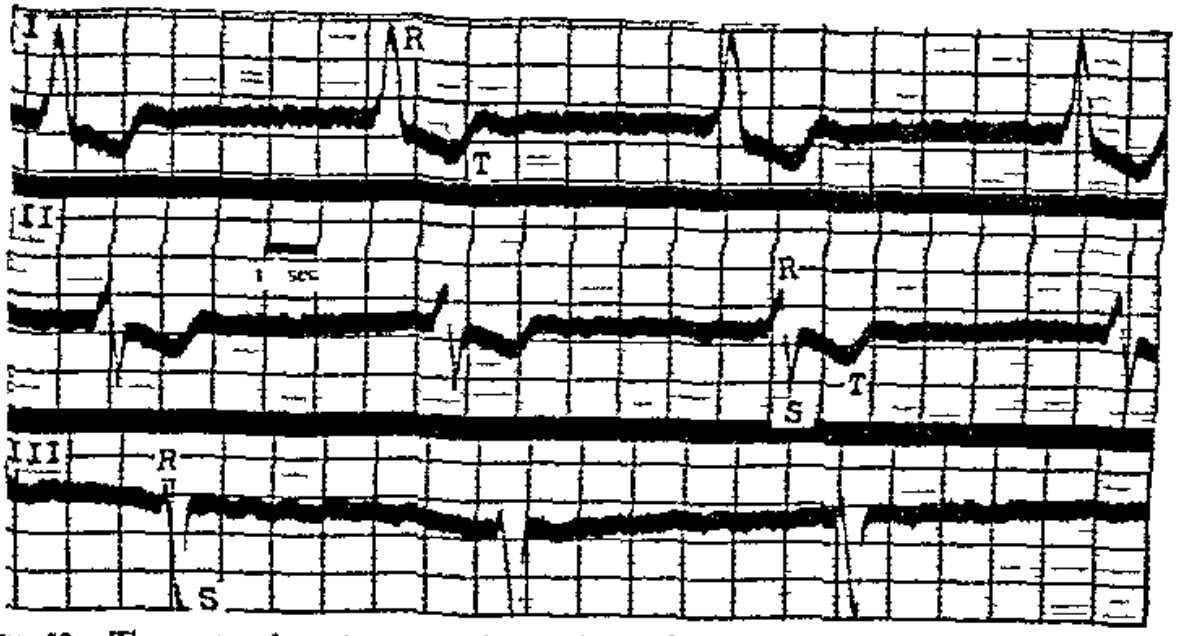


FIG 52.—*The ventricular rate is very slow The rhythm is irregular from auricular fibrillation in complete heart block* L A D The T waves are inverted in leads I II and III

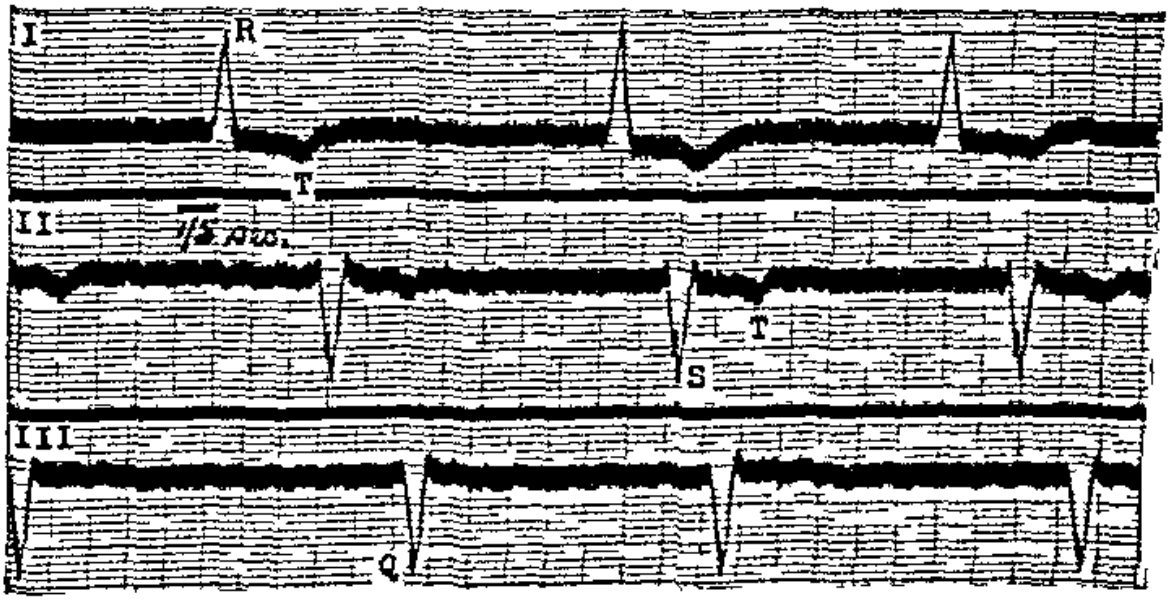


FIG 53.—The ventricular rate is very slow The rhythm is irregular from auricular fibrillation L A D The T waves are inverted in leads I and II

normally unless in the presence of auricular tachycardia (Fig 51) or auricular fibrillation (Fig 52) which may be resembled by very slow fibrillation (Fig 53) which is the commoner condition

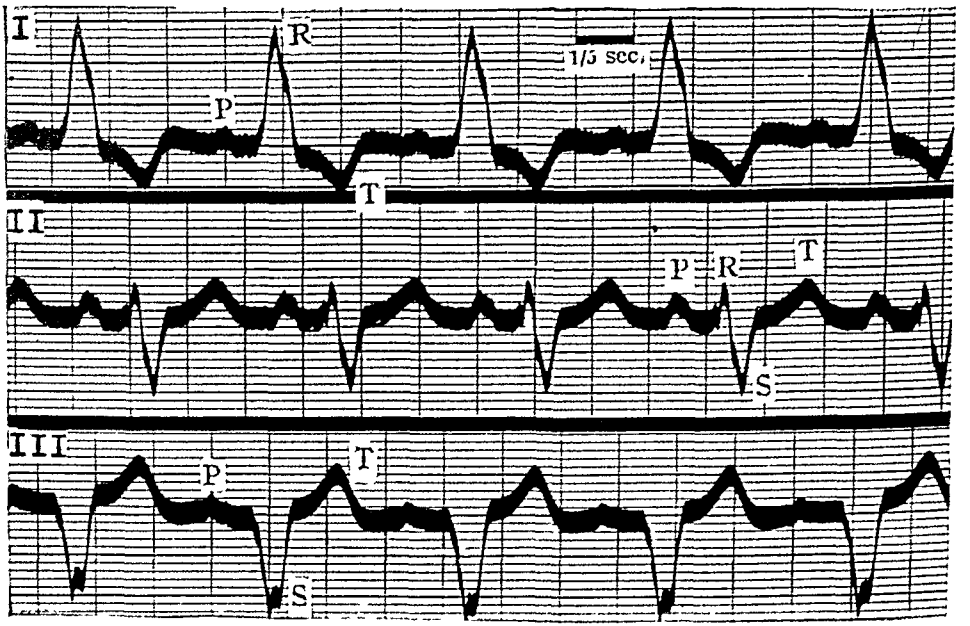


FIG. 54.—The rate is normal. The rhythm is regular. The P–R period is slightly prolonged. L.A.D. The QRS complexes are wide and the T waves are written in the opposite direction to the QRS.

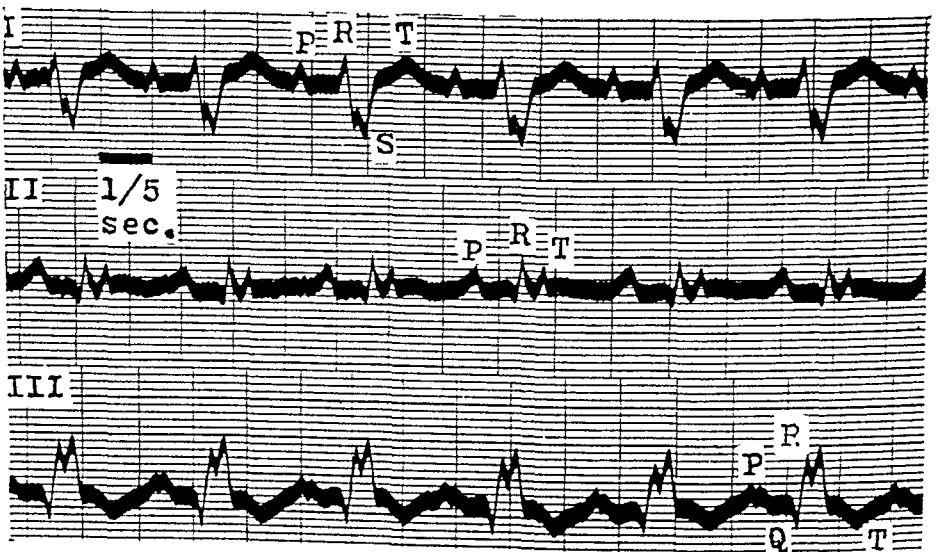


FIG. 55.—The rate is normal. The rhythm is regular. The P–R period is normal. R.A.D. The QRS complexes are wide and the T waves are written in the opposite direction to the QRS.

Bundle branch block

The auricular wave is normal in this condition unless there is auricular fibrillation. The P–R period is often prolonged, but it is short in the Wolff-Parkinson-White syndrome The QRS complex is wide and exceeds the normal interval of

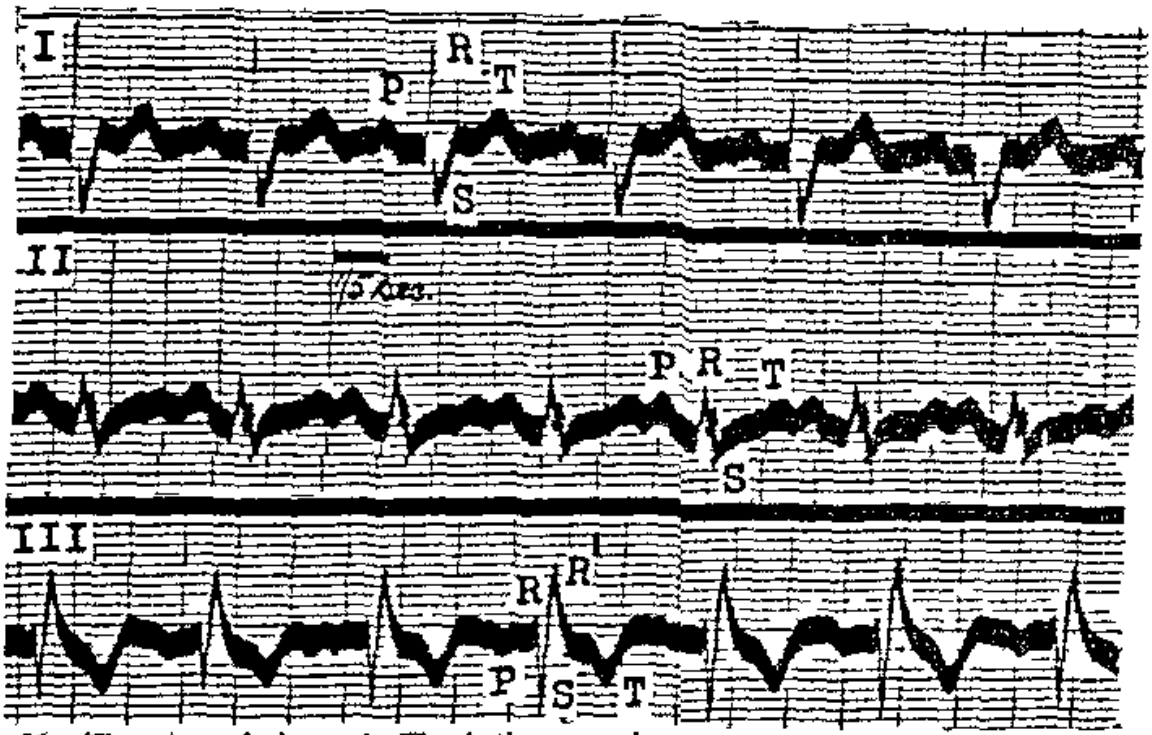


FIG 56 —The rate is a little rapid The rhythm is regular The P–R period is prolonged R A D The S wave is deep and broad in leads I and II The T wave is inverted in lead III

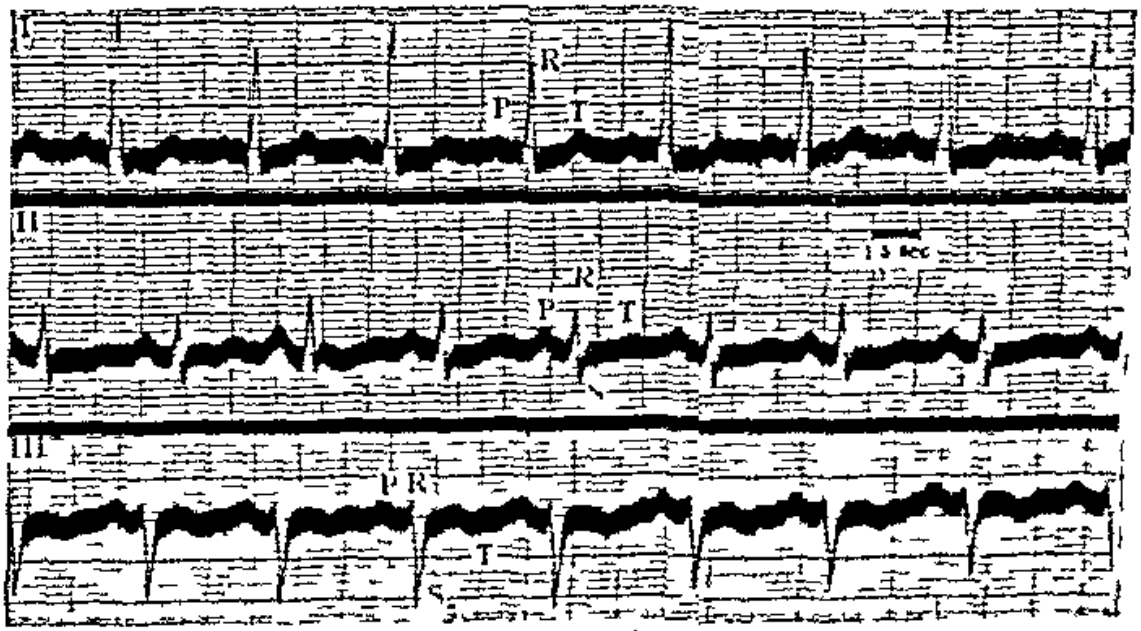


FIG 57 —The rate is normal The rhythm is regular The P–R period is normal L A D The voltage of alternate QRS complexes in lead I varies The T wave is low in leads I and II and inverted in lead III

0·1 second ; it shows notching of either its ascending or its descending limb. The T waves that follow are known as secondary T waves and are usually large and written in the opposite direction to the QRS deflection.

In *left bundle branch block* (Fig. 54) the main QRS wave is directed upwards in lead I and downwards in lead III.

In the *uncommon* kind of *right bundle branch block* (Fig. 55) the main QRS wave is directed downwards in lead I and upwards in lead III.

In the *common* kind of *right bundle branch block* (Fig. 56) the curve is characterized by a deep S wave as component of a wide QRS complex in lead I and in

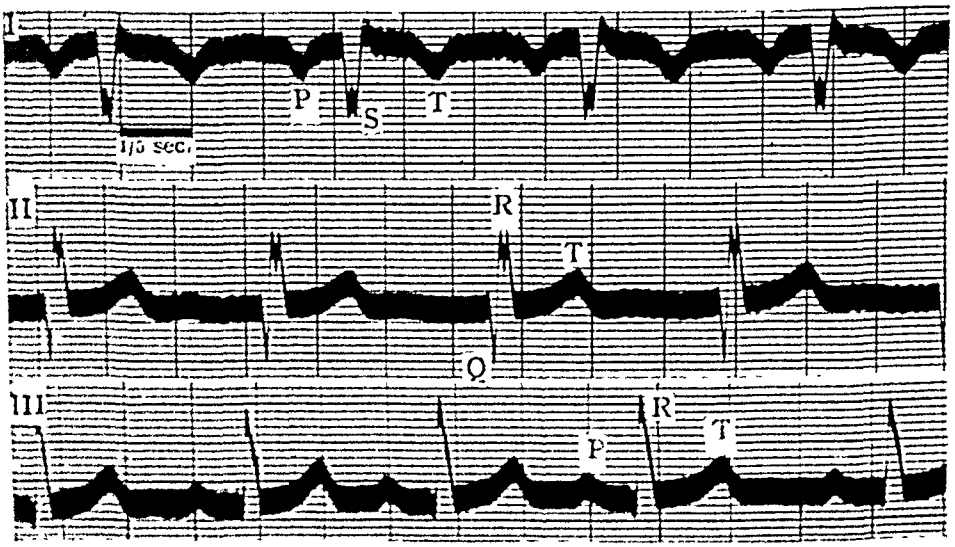


FIG. 58.—The rate is normal. The rhythm is regular. The P–R period is normal. The P, QRS, and T waves in lead I are inverted. The P wave in lead II is indistinct, and the Q wave is deep. The QRS complexes are wide.

lead II as a rule. The T wave is upright in leads I and II and usually inverted in lead III. The QRS wave in lead III is also wide, and although it may be upright it is more often directed downwards when characteristically a deep Q wave is present. The chest leads CR_1 and CR_2 are useful to test the presence of right and left bundle branch block respectively.

FAULTY HEART CONTRACTION

Electrical alternans

The electrocardiogram in pulsus alternans is not necessarily abnormal, except in showing left ventricular preponderance, and it seldom shows variation in electrical potential in alternate beats. Such electrical variation, however, may be found (Fig. 57) in the absence of obvious heart disease.

CONGENITAL *HEART DISEASE*

Apart from dextrocardia the electrocardiogram of congenital heart disease does not differ from that obtained in acquired disease The voltage is often high and

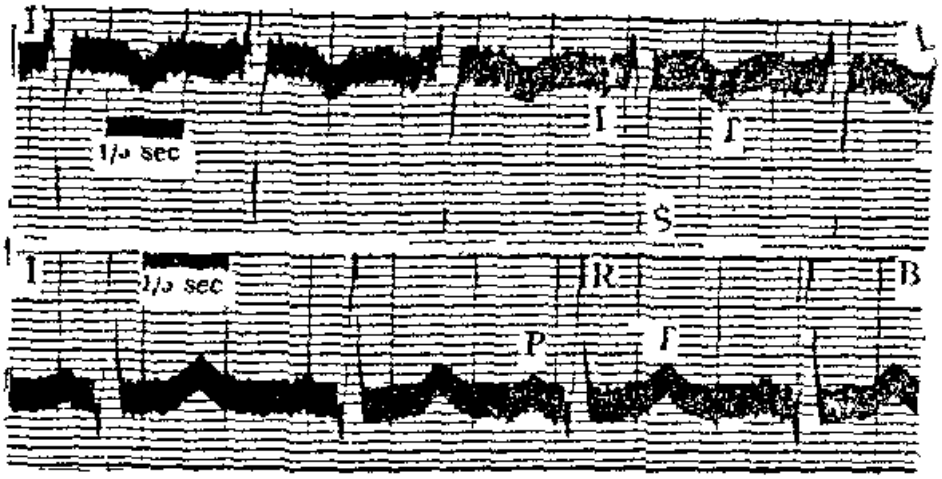


FIG 59.—The P QRS and T waves are inverted in lead I in (A) they are upright in (B) where the right and left arm electrodes were interchanged

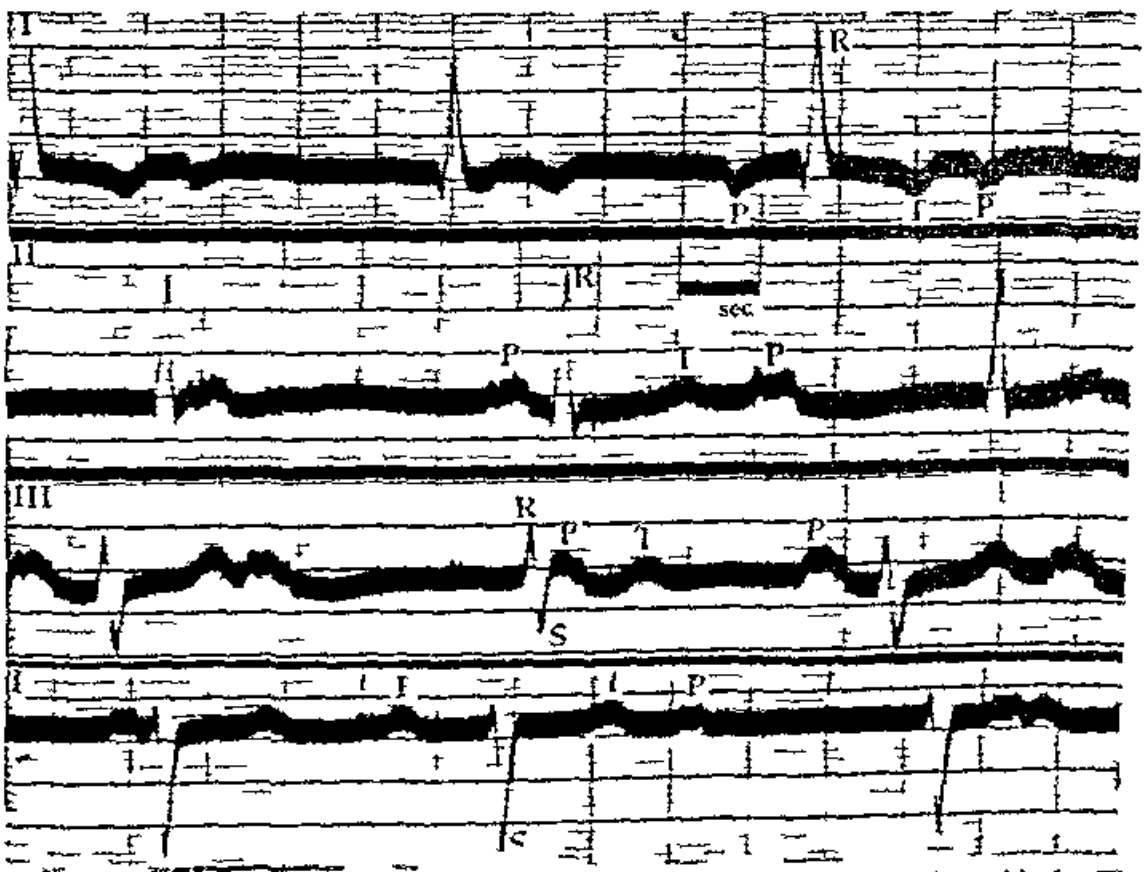


FIG 60.—The ventricular rate is slow The rhythm is irregular from complete heart block The P and T waves in lead I are inverted from congenital dextrocardia but the R wave is upright because of R.A.D. from pulmonary stenosis. The lower tracing was recorded with the right and left arm electrodes reversed

faults of conduction are sometimes present in the form of auriculoventricular or bundle branch block. Since hypertrophy of the right heart is common in congenital disease, right axis deviation and right ventricular preponderance are

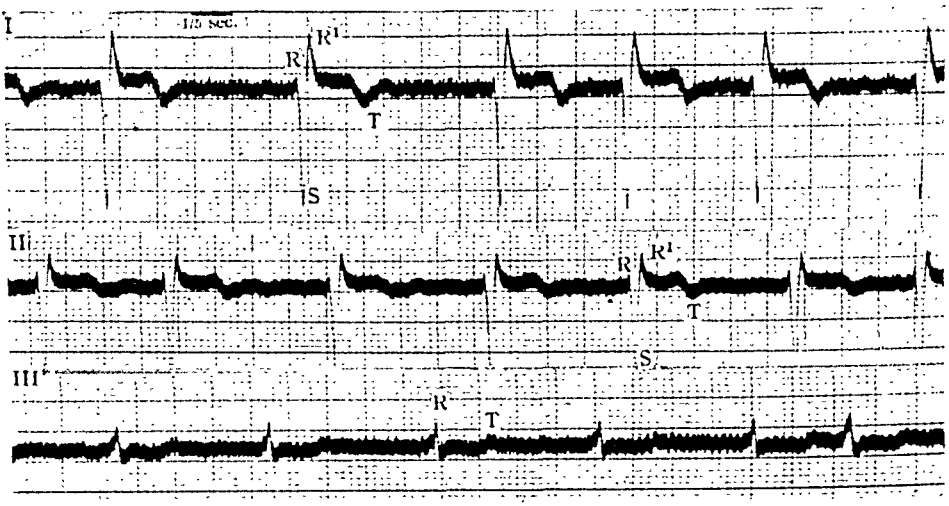


FIG. 61.—The rate is normal. The rhythm is irregular from auricular fibrillation. The QRS complex and the T waves are reversed in leads I and II, and the R–T segments are raised as a mark of left heart preponderance, in congenital dextrocardia.

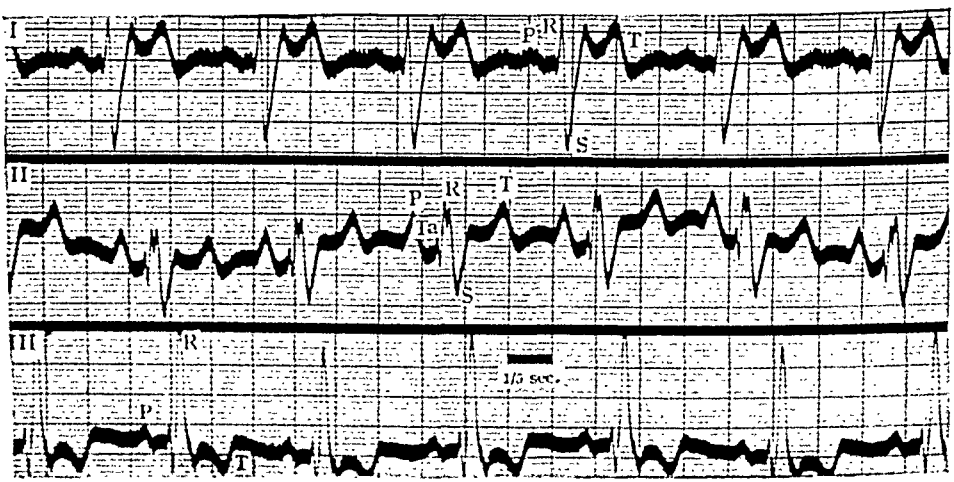


FIG. 62.—The rate is normal. The rhythm is regular. The P–R period is normal. R.A.D. The QRS complexes are wide and the T wave is directed in the opposite direction to the QRS.

natural cardiographic findings. Thus, a deep S wave in lead I, and inversion of the T waves in leads II and III, and in CR_1, are characteristic changes in many congenital heart defects, and cannot by themselves have diagnostic value, so that differentiation has to be made on clinical grounds aided by radiological examination.

Congenital dextrocardia

The limb lead cardiogram (Fig 58) shows inversion of the P QRS and T waves in lead I, and an interchange of the tracings in leads II and III An orthodox curve in lead I is obtained by reversing the right and left arm terminals (Fig 59)

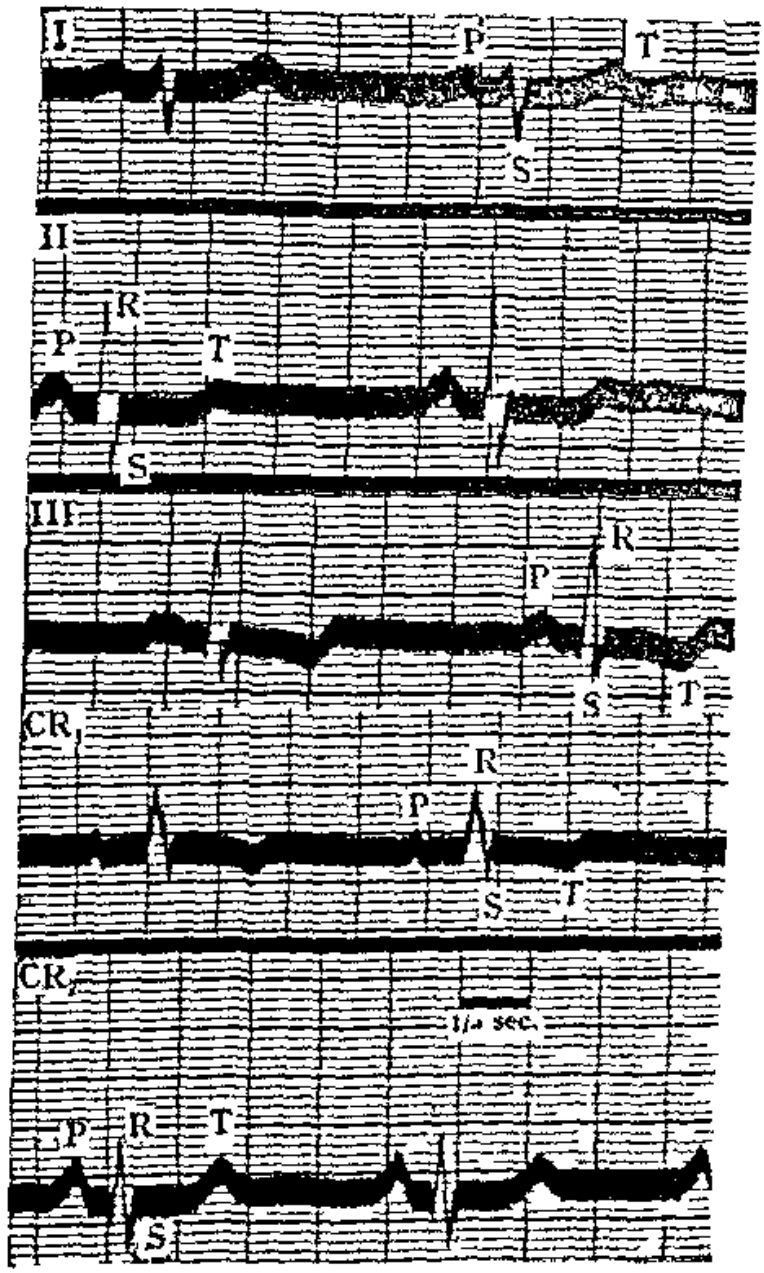


FIG 63.—The rate is normal The rhythm is regular The *P R* period *is normal* R A D The P wave is broad The T wave is inverted in leads III and CR_t and diphasic in lead II

The displaced heart is susceptible to congenital and acquired disease When a congenital defect is added, the R wave in lead I again appears upright as an expression of right ventricular preponderance (Fig 60) whereas in hypertension, the downward projection of the QRS becomes even more noticeable with elevation of the R–T segment (Fig 61)

Coarctation of the aorta

The cardiogram of coarctation may often be physiological, or it may show left axis deviation, or early changes due to left ventricular preponderance, especially in the presence of aortic incompetence.

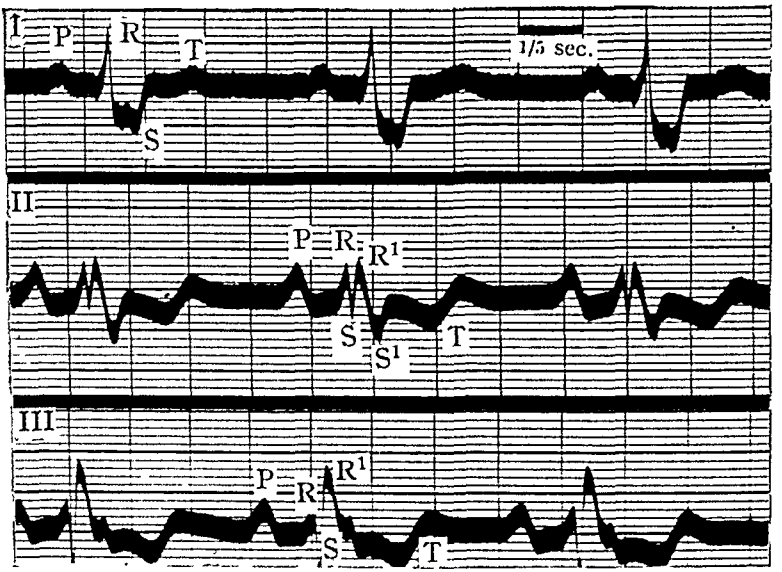


FIG. 64.—The rate is normal. The rhythm is regular. The P–R period is normal. R.A.D. The QRS complexes are wide and the T wave is written in the opposite direction to the QRS.

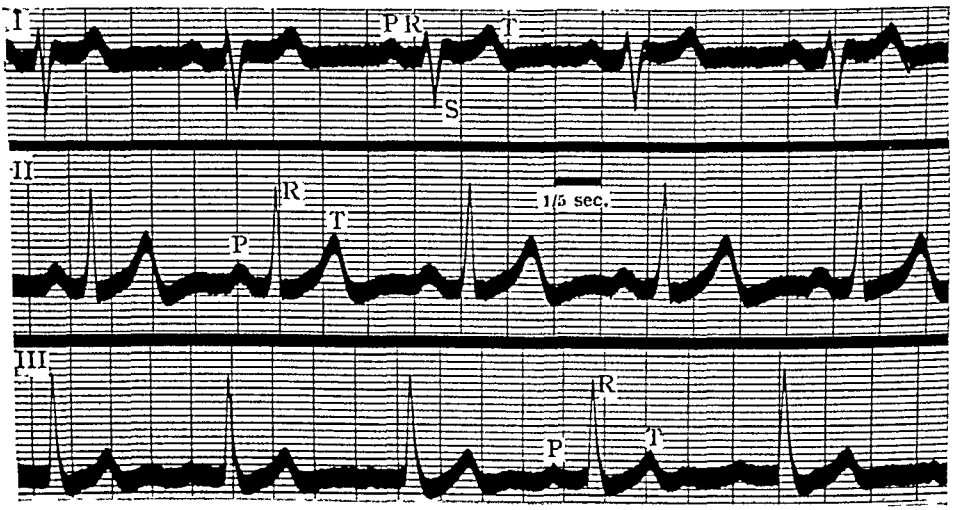


FIG. 65.—The rate is normal. The rhythm is regular. The P–R period is normal. R.A.D. The T wave is upright in all leads.

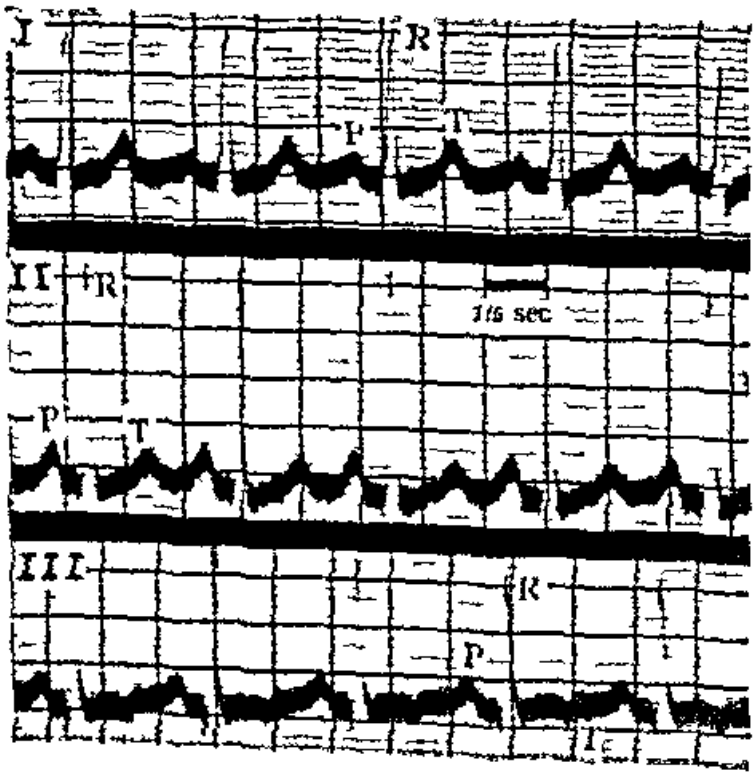


FIG 66 —The rate is a little rapid and the rhythm is regular (sinus tachycardia) The P R period is normal No deviation of the electrical axis. The T wave is inverted in lead III

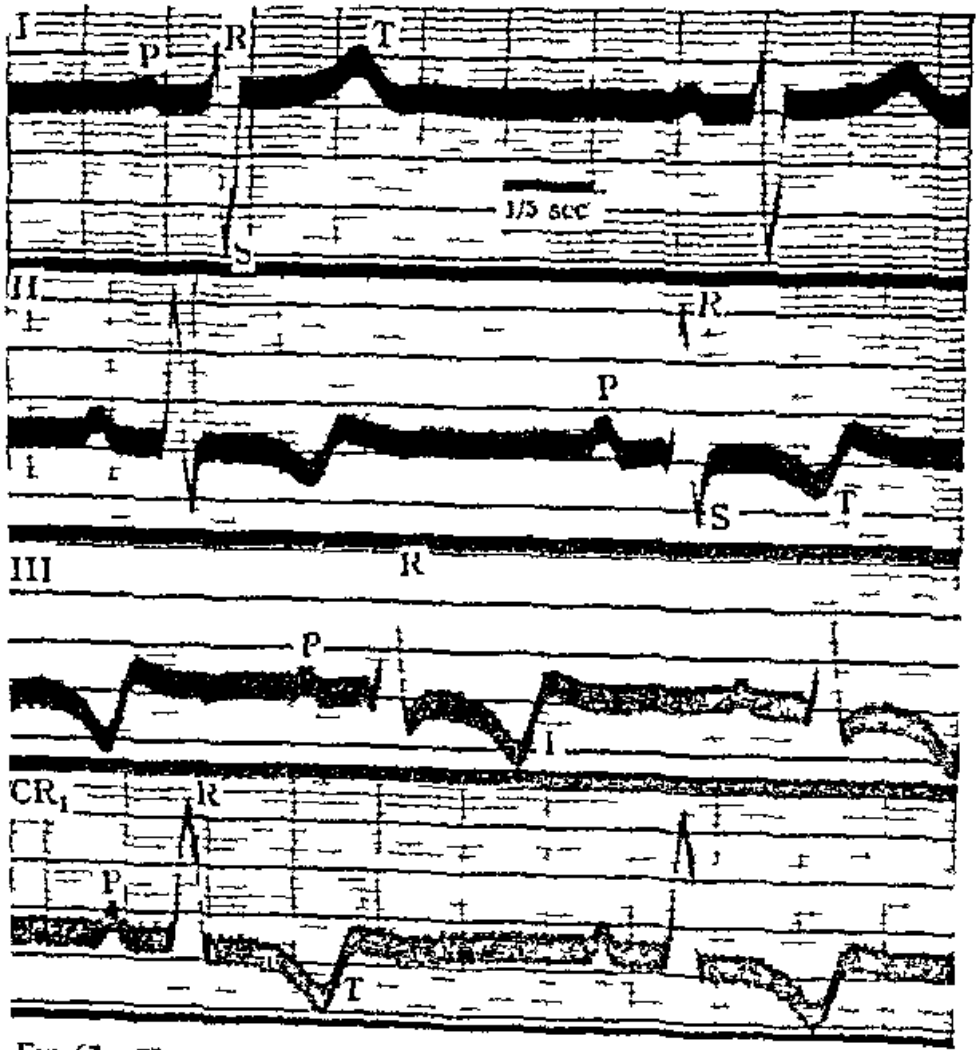


FIG 67 —The rate is a little slow The rhythm is regular The P R period is normal R A D There is no S wave in CR_1 and the T is inverted in leads II III and CR_1

Subaortic stenosis

The differentiation of congenital subaortic stenosis from acquired aortic stenosis is notably difficult on clinical grounds. The congenital nature of the lesion receives confirmation from the finding of right axis deviation (Fig. 62) which may be present sometimes.

Auricular septal defect

The cardiogram of auricular septal defect is usually abnormal and it often shows deviation of the electrical axis to the right, bundle branch block, or right heart preponderance expressed by inversion of the T wave in leads II, III and CR_1 (Figs. 63 and 64).

Ventricular septal defect

As a rule, the changes in the T waves characteristic of right ventricular preponderance are absent in this condition unless accompanied by other congenital defects, and right axis deviation or notching of the QRS are usually the only abnormal cardiographic changes (Fig. 65).

Patent ductus arteriosus

The cardiogram of patent ductus arteriosus is physiological (Fig. 66). An abnormal tracing in congenital heart disease makes it unlikely that patent ductus arteriosus is the lesion which produces the clinical signs.

Pulmonary stenosis and pulmonary atresia

This congenital anomaly of the pulmonary opening, existing either alone as pulmonary stenosis, or in association with a defect of the ventricular septum (Fallot's syndrome), produces a characteristic cardiogram (Fig. 67) showing the effects of right axis deviation combined with those of right ventricular preponderance. Thus, the S wave is deep in lead I, and the T wave is inverted in leads II, III and CR_1. The R wave is greater than S in CR_1, and the reverse is true in CR_7.

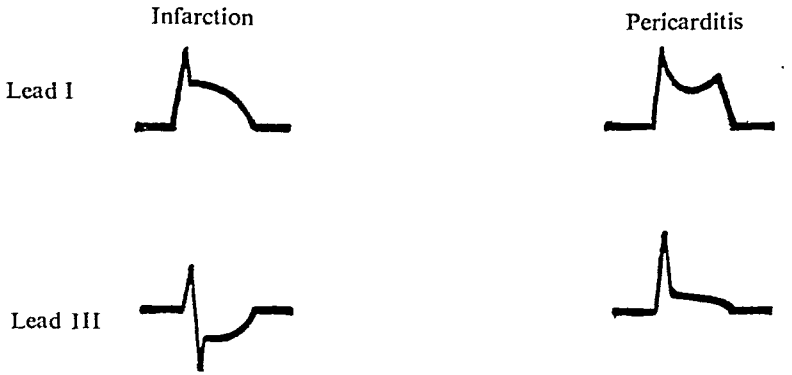


FIG. 68.—Direction and form of the R–T deviation in leads I and III in cardiac infarction and in acute pericarditis.

PERICARDIAL DISEASE

The abnormal cardiogram identified with pericardial disease is not the outcome of increased intrapericardial pressure but is the result of direct extension into the heart muscle of the disease which is affecting the pericardium

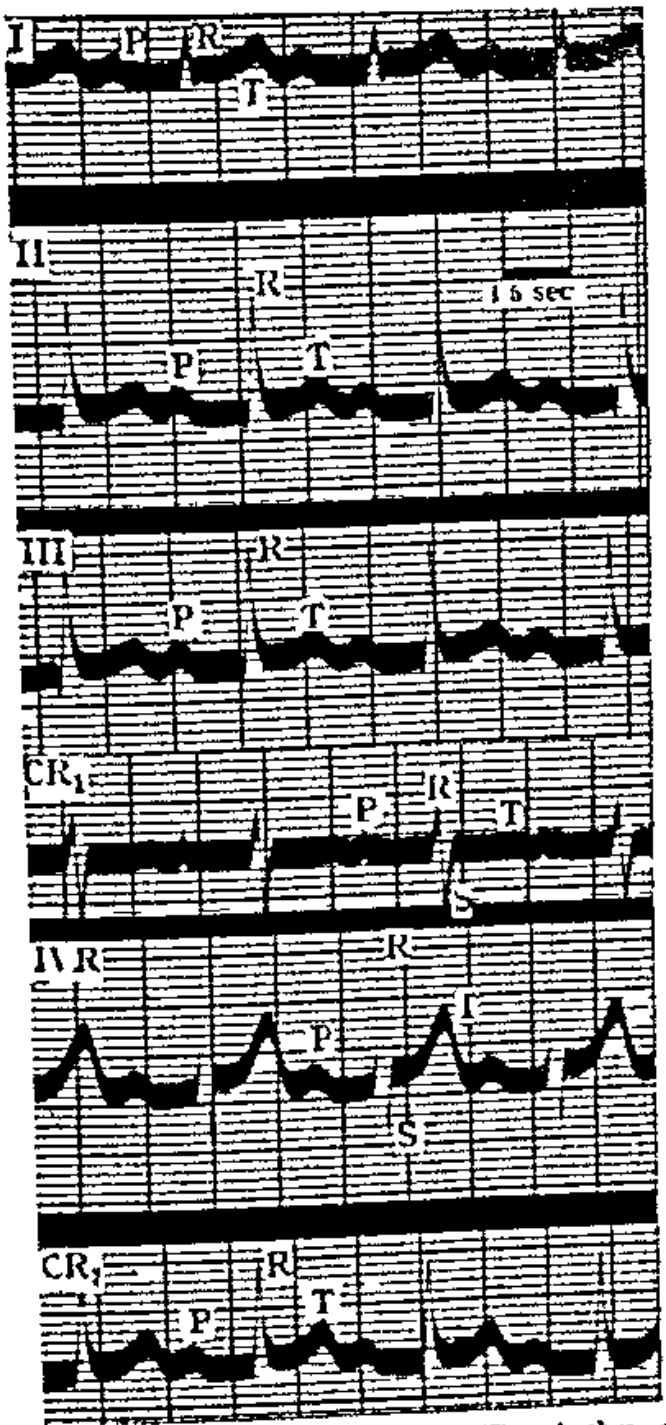


FIG 69.—The rate is normal The rhythm is regular The P R period is prolonged No axis deviation The R T segment is raised in all leads.

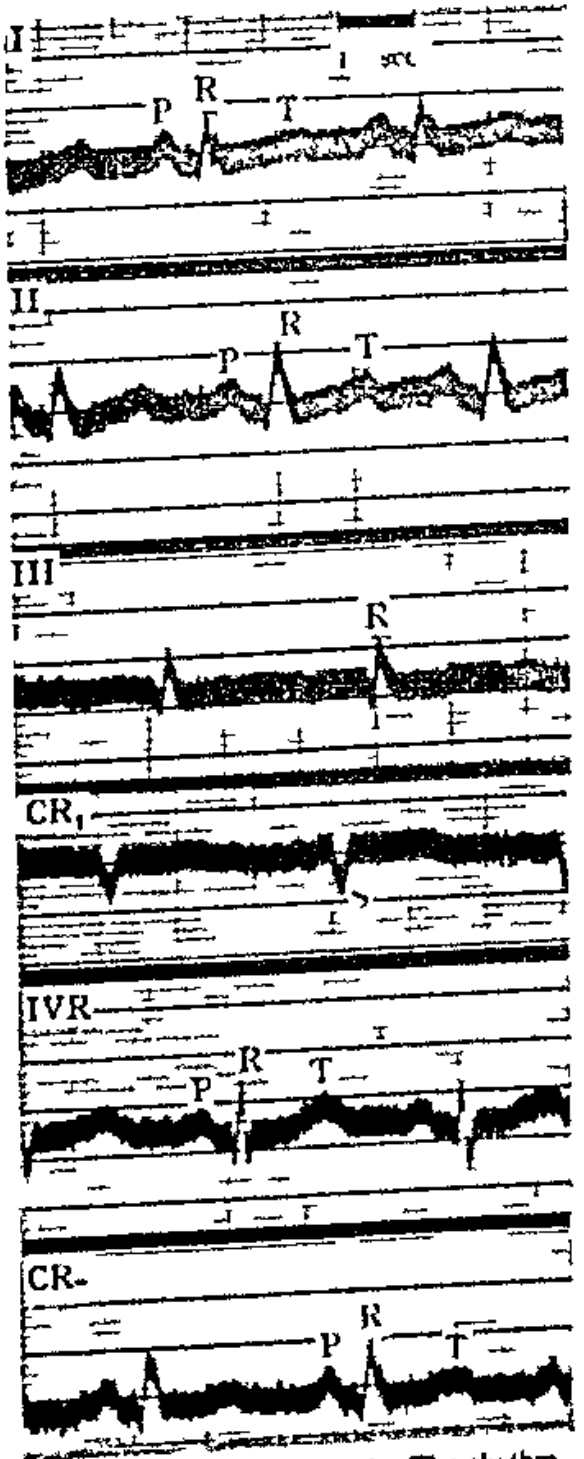


FIG 70.—The rate is normal The rhythm is regular The P R period is normal No axis deviation The QRS complexes are a little wide and of low voltage The T waves, though low in some leads, are upright in all

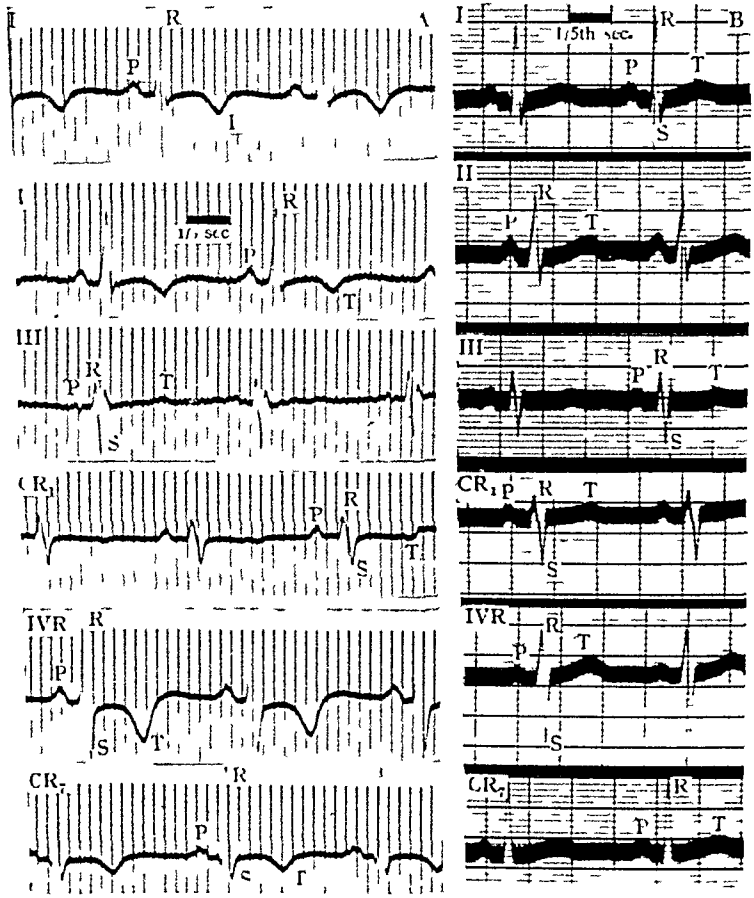


FIG. 71.—The rate is normal The rhythm is regular. The P–R period is normal. L.A.D. The T waves in (A) are inverted in leads I, II, CR_1, IVR, and CR_7, but are upright in (B) taken three weeks later

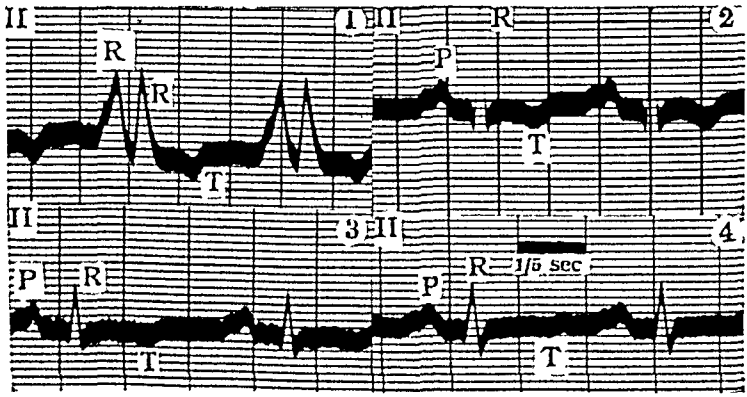


FIG. 72.—Four examples of broadening of the P waves with a shelf or step appearance taken from patients with pericardial disease.

The changes in the cardiogram of *acute pericarditis* are confined in the early stages to the R–T segment Like those of cardiac infarction they are short lived and are followed by inversion of the T wave Unlike cardiac infarction the form

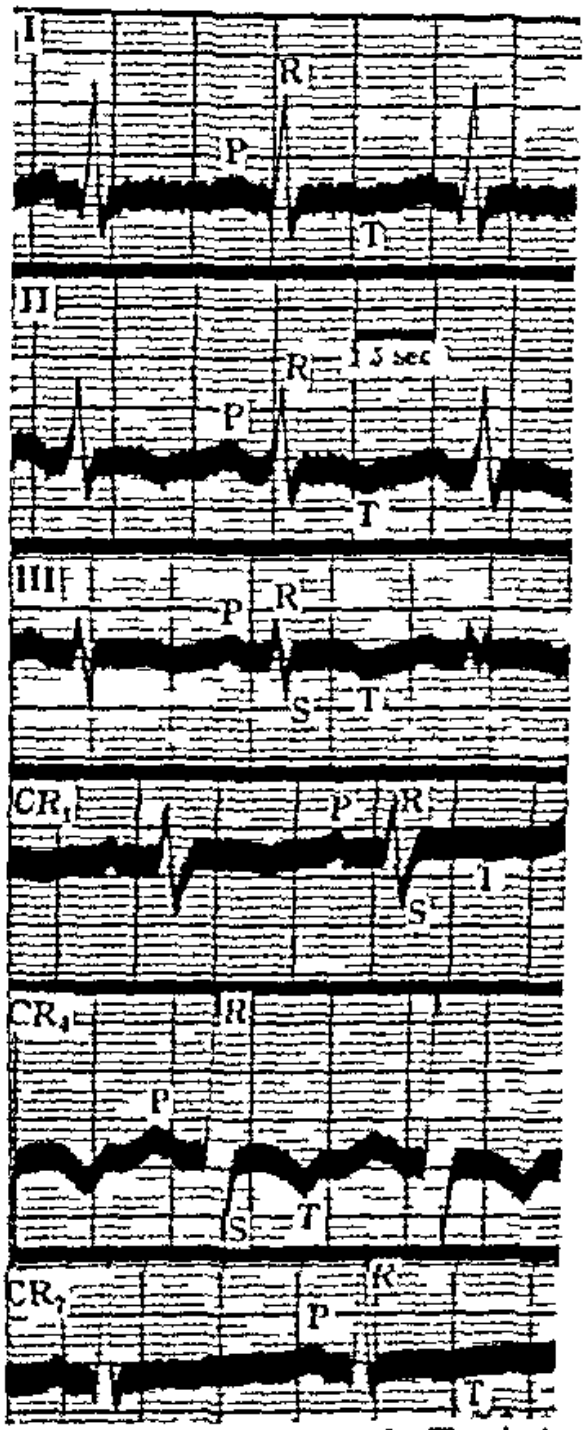


FIG 73 —The rate is normal The rhythm is regular The P R period is normal No axis deviation The T wave is low in leads I and CR and inverted in II III CR and CR_4

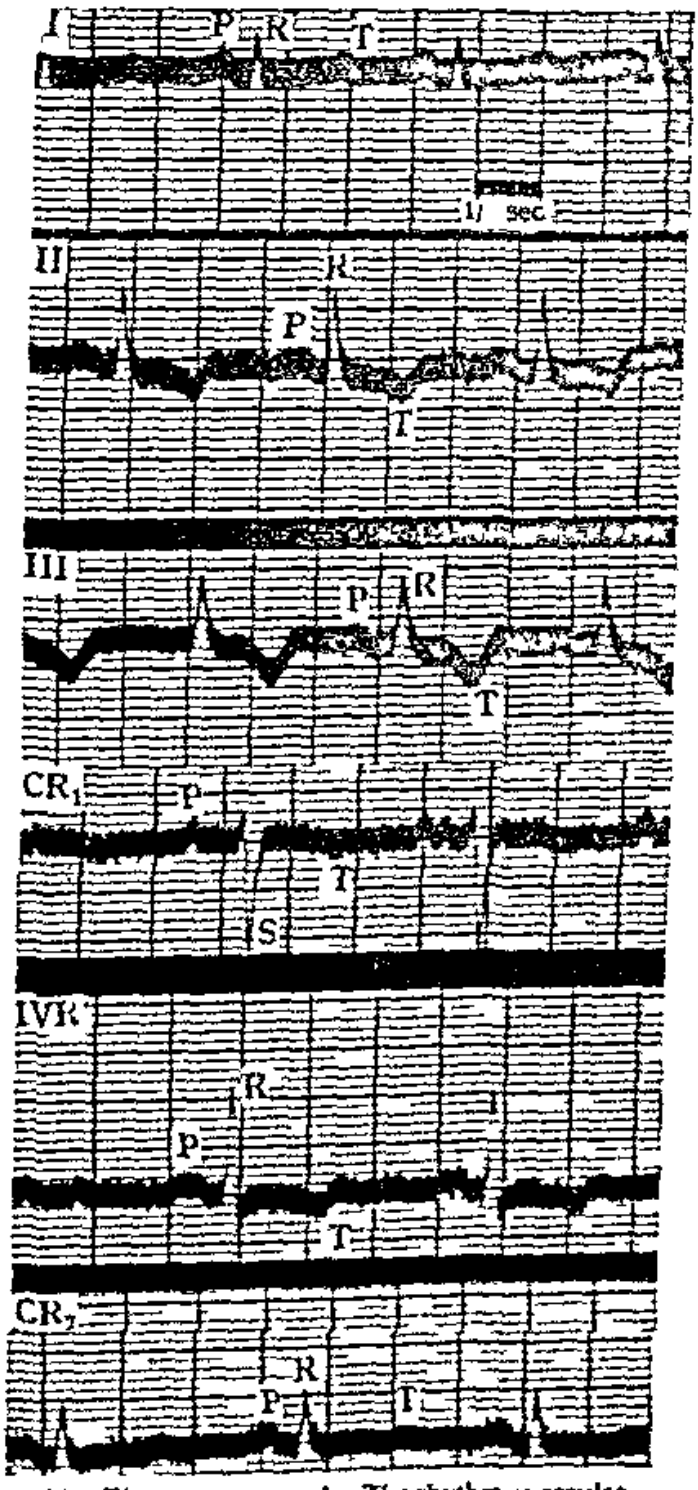


FIG 74 —The rate is normal The rhythm is regular The P R period is normal No axis deviation The P wave is low and a little broad The T wave is upright in leads I and CR_1 and inverted in the others

and direction of the R–T deviation is different. Thus, the elevated R–T_1 of cardiac infarction shows coving with the convexity of the curve upwards, while the elevated R–T_1 of pericardial disease shows an upward concavity. Again, the reciprocal relationship between changes in leads I and III in cardiac infarction, namely elevation of the R–T segment in one lead and depression in another, is absent in pericarditis (Figs. 68 and 69).

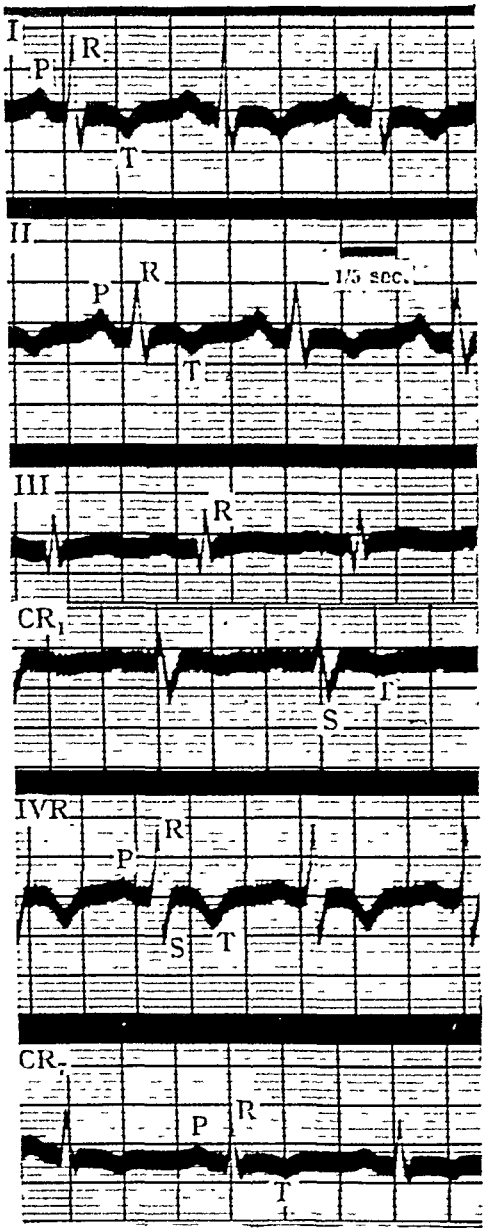


FIG. 75.—The rate is normal. The rhythm is regular. The P–R period is normal. No axis deviation. The P wave is a little wide. The T wave is low in lead III and inverted in all other leads.

While the duration of the R–T changes is no longer than a few days, the deformity of the T wave which follows will last or disappear according to the progress of the underlying myocardial injury, and it does not depend on the presence or absence of pericardial fluid.

The cardiogram shown in Fig. 70 is not appreciably altered in spite of great distension of the pericardial sac by haemorrhagic effusion. The abnormal tracing in Fig. 71 was obtained when pericardial effusion had formed in acute pericarditis and it was normal three weeks later when the fluid was absorbed. Again, the tracing in Fig. 73 was obtained from a patient with acute pericarditis in whom no effusion occurred.

The cardiogram of constrictive pericarditis shows lasting changes which are not abolished by time or even by a successful operation. In such a tracing the voltage is sometimes low; the rhythm is often irregular from auricular fibrillation even in young patients; the P wave is usually broad and often demonstrates a shelving effect in lead II (Fig. 72); the Q wave is never prominent, and the R–T segment seldom shows any deviation. Changes in the T wave make it convenient to consider them in two groups, although some fall outside these.

In the first type (Fig. 74) the T wave is inverted in leads III, II, CR_1, and in CR_1 or IVR, but is upright in I and CR_7.

In the second type (Fig. 75) the

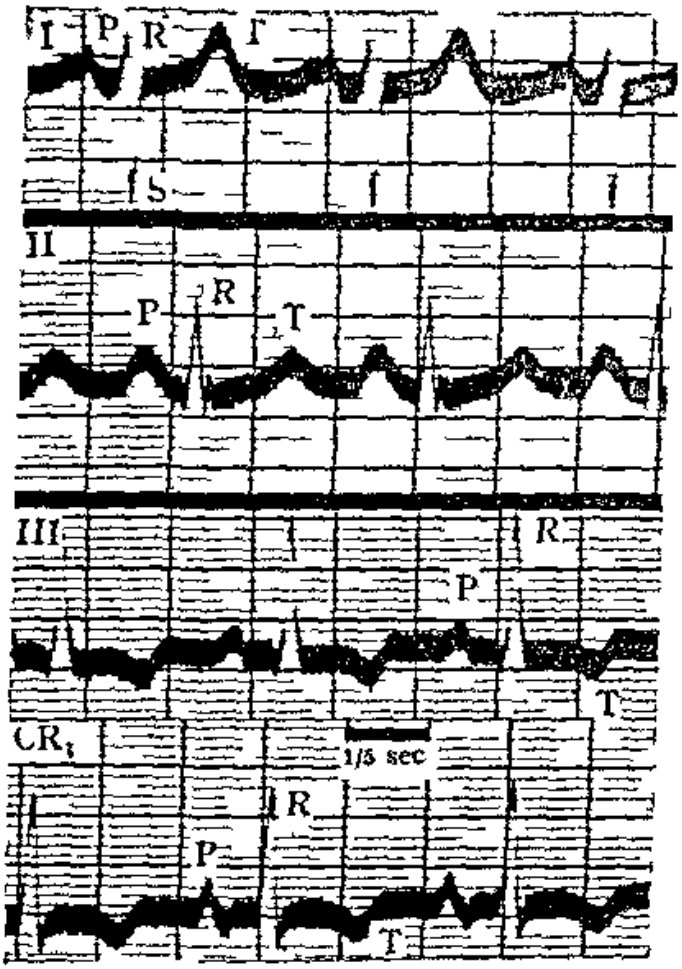


FIG 76.—The rate is normal. The rhythm is regular. The P R period is normal. R A D. The P wave is wide in lead I, tall in II, and diphasic in CR. The T wave is inverted in III and in CR_1 where the S wave is absent.

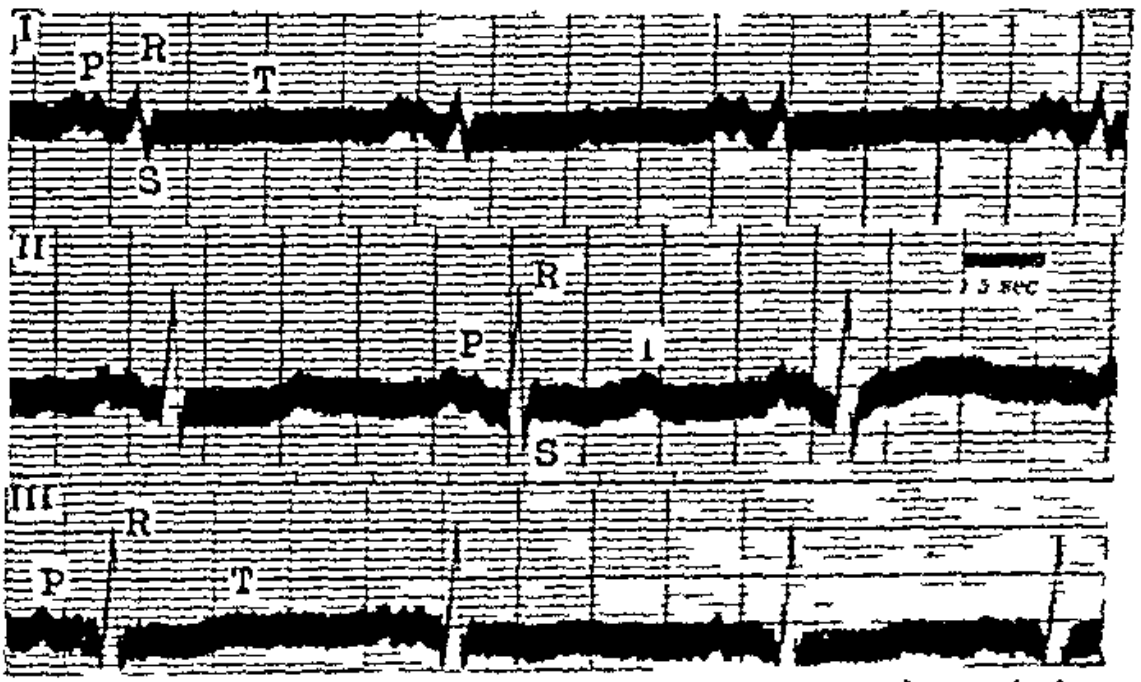


FIG 77.—The rate is normal. The rhythm is regular. The P R period is normal. No axis deviation. Small voltage in lead I. The P waves are broad and bifid. The T waves are low from digitalisation.

T wave is inverted in leads I and II, and in CR_7 usually to a greater extent than in CR_4. The T wave in CR_1 is either low or inverted.

MITRAL STENOSIS

In patients with early mitral stenosis there may not be any changes in the electro-

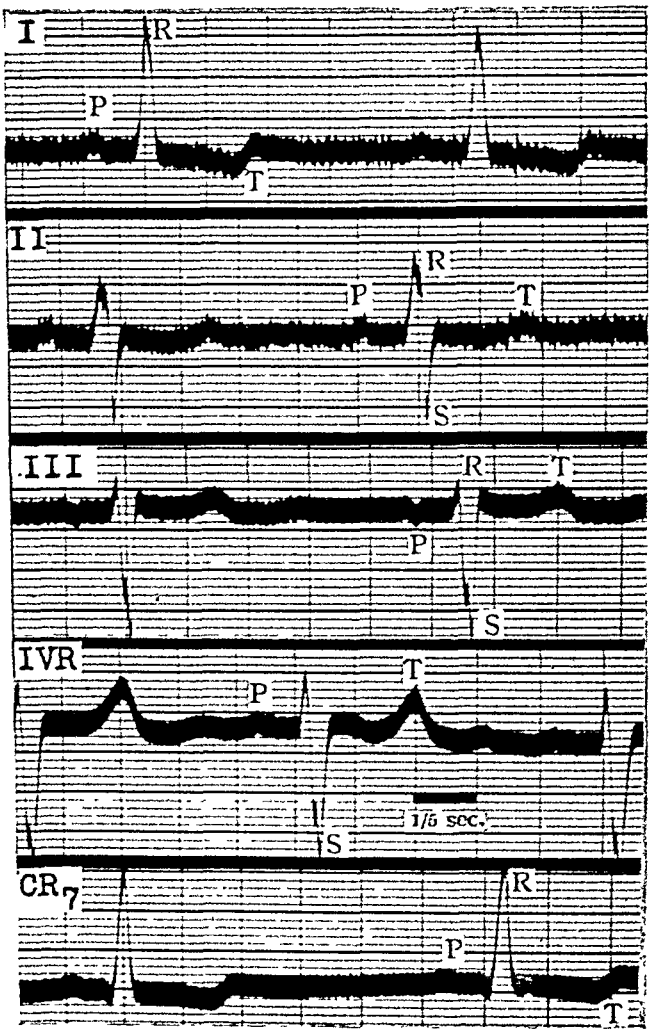


FIG. 78.—The rate is a little slow and the rhythm is regular (sinus bradycardia). The P–R period is full. L.A.D. The T wave is inverted along with R–T depression in leads I and CR_7.

cardiogram so that this test cannot help in diagnosis at the commencement of the illness and when the clinical signs may be equivocal. As the condition progresses the P wave becomes broader and often bifid especially in lead I; in CR_1 the P may be tall and spiky. When the condition is well developed there

is right axis deviation but the voltage in lead I is usually low (Figs 76 and 77) Inversion of the T wave in leads CR_1 II and III is less noticeable in mitral stenosis than in other instances of right heart enlargement and failure the presence of such changes suggests the addition of auricular septal defect to mitral stenosis (Lutembacher s syndrome) or that pulmonary incompetence is a prominent feature When tricuspid stenosis is added the cardiographic signs typifying mitral stenosis are more likely to be present

The tracing of mitral stenosis frequently shows auricular fibrillation and the effects of digitalis therapy

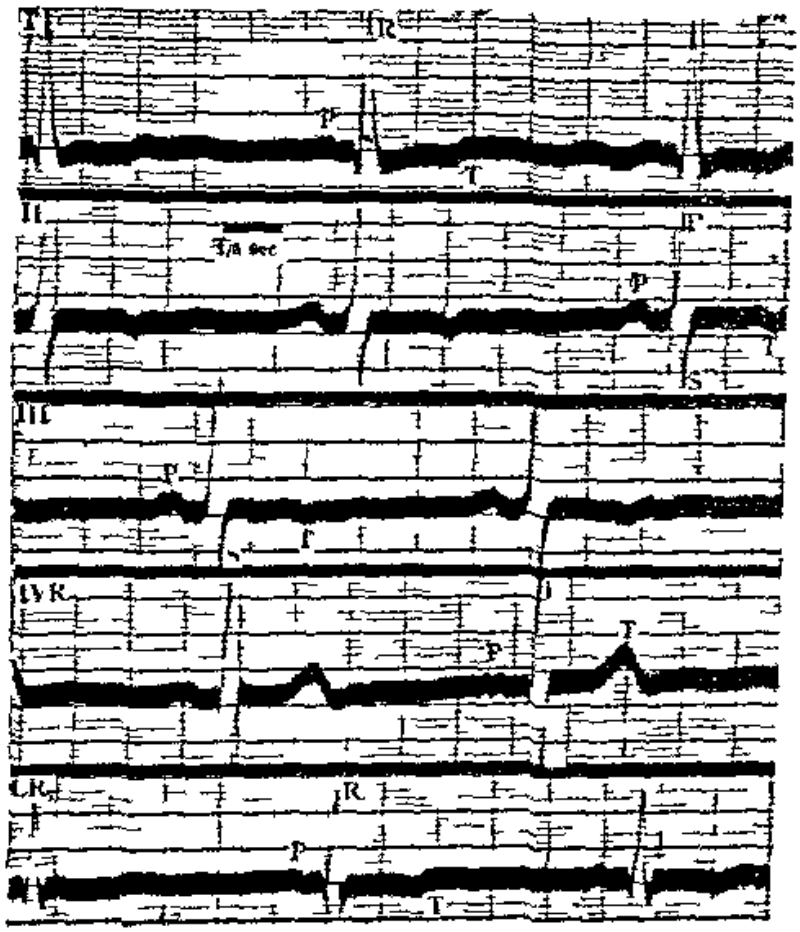


FIG 79 —The rate is a little slow and the rhythm is regular (sinus bradycardia) The P R period is normal No axis deviation The T wave is inverted in leads I II III and CR

AORTIC VALVULAR DISEASE

The cardiogram of aortic incompetence (Fig 78) is similar to the one found in many cases of left ventricular enlargement from hypertension There is left electrical axis deviation and inversion of the T wave in leads I and CR_7 should the T wave be inverted in IVR as well the change is not as a rule as great as that in CR_7

Sometimes the inversion of the T wave in limb leads may involve leads II and III in addition to or without inversion of the T in lead I (Fig. 79)

The same changes are found in aortic stenosis (Fig. 80) and in addition a characteristic steep ascent of the T wave may be observed at the termination of a more gradual R–T depression.

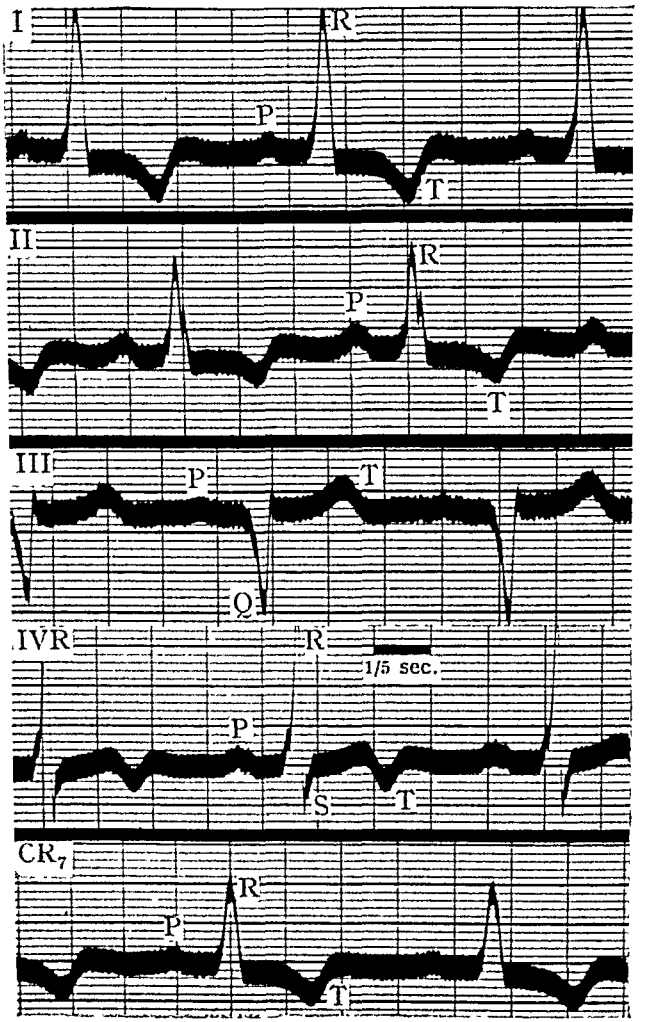


FIG. 80.—The rate is normal. The rhythm is regular. The P–R period is prolonged. L.A.D. The T wave is inverted in leads I, II, IVR, and CR_7; the inversion in CR_7 is greater than in IVR. The R–T segment is depressed in leads I, II and CR_7.

CARDIAC INFARCTION

The early cardiographic changes in cardiac infarction are confined to the R–T segment; they appear within an hour or less of the attack and may last for several days before giving way to the typical T wave deformity, when the R–T segment may have returned to its isoelectric level. The R–T deviation (Pardée sign) is

a coving effect with its convexity upwards in lead I and an opposite or discordant effect in lead III in the anterior type (Fig 81) which differentiates the change from that found in acute pericarditis In posterior infarction the changes are

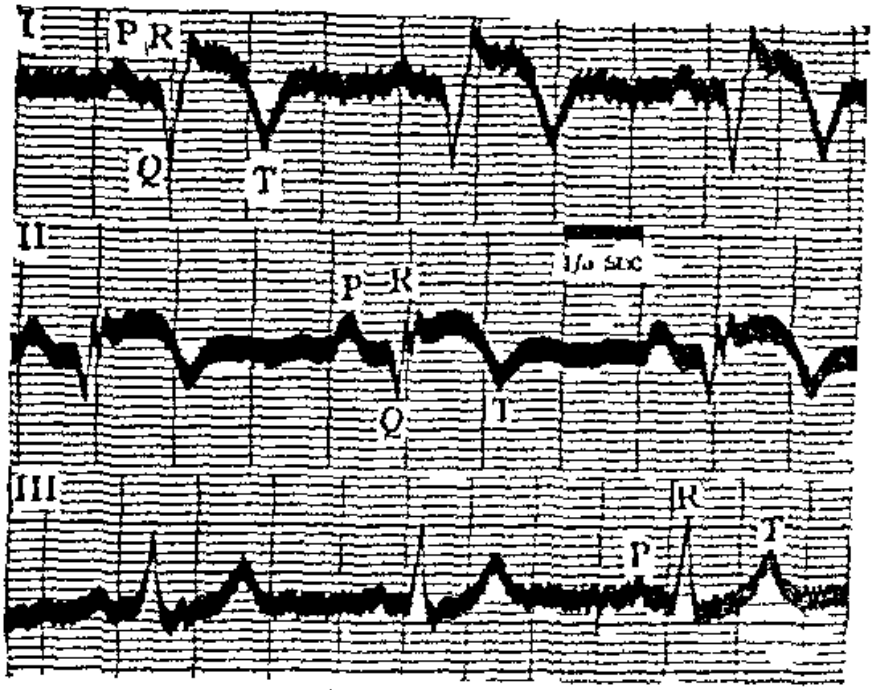


FIG 81 —The rate is normal The rhythm is regular The P R period is normal The Q wave is prominent and the T wave is inverted with R T deviation which is best seen in leads I and II

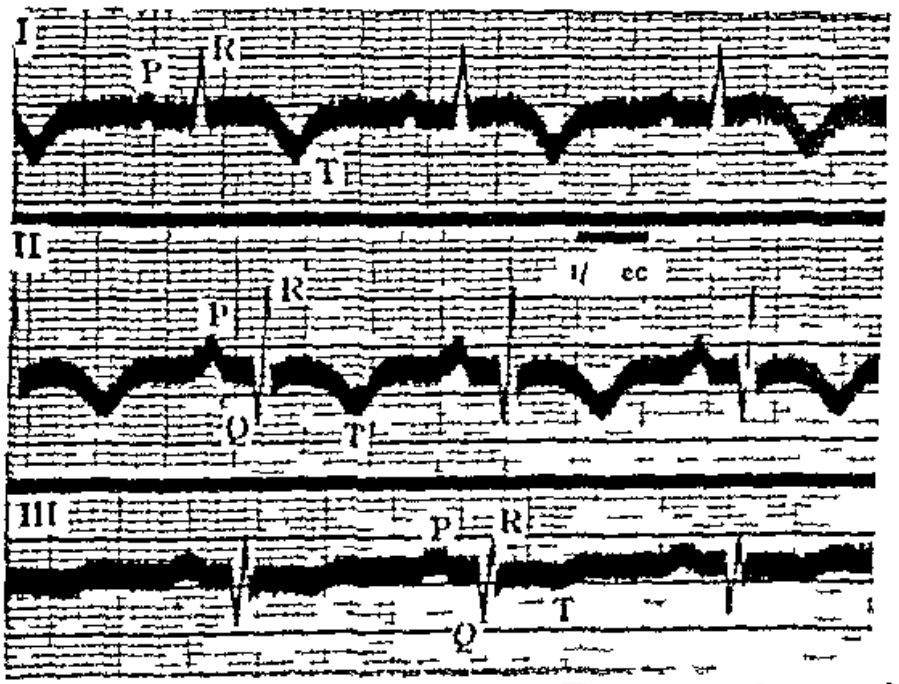


FIG 82 —The rate is normal The rhythm is regular The P R period is normal No axis deviation The T wave is inverted in leads I and II and slightly inverted in III

in the reverse direction In anterior cardiac infarction the R–T changes may be slight at the start and the T wave in lead I may remain upright although a little low , in this circumstance lead CR_4 or IVR gives invaluable aid to diagnosis ,

here the R–T segment might be obviously raised and a Q wave present. Soon the T wave becomes inverted.

The later cardiographic changes are described here according to whether the anterior or posterior part of the heart is affected.

Anterior infarction.—The changes conform to a T_1 pattern described by Parkinson and Bedford where the T wave is inverted in lead I and often in lead

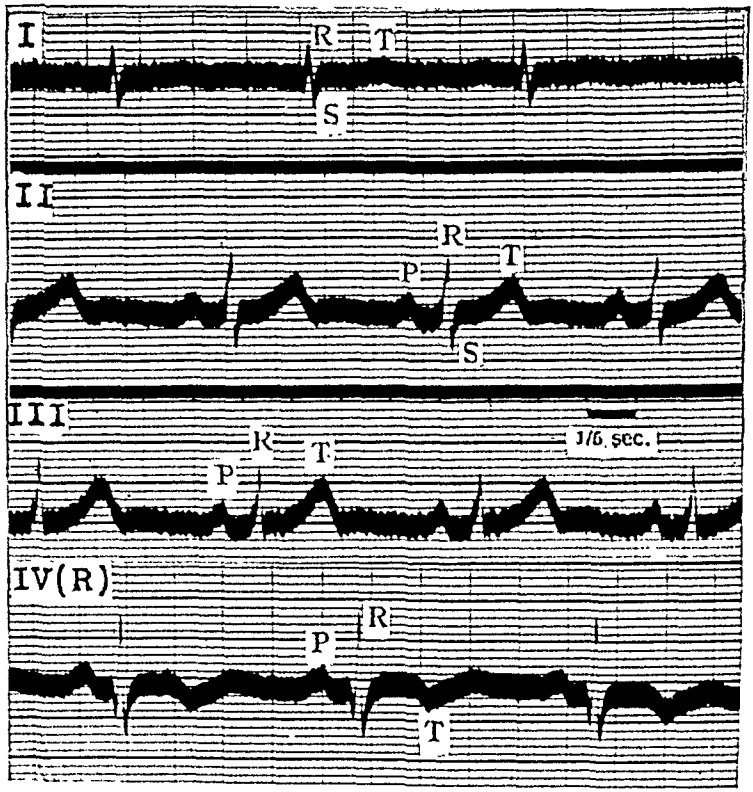


FIG. 83.—The rate is normal. The rhythm is regular. The P–R period is normal. No axis deviation. *The T wave though low is upright in lead I, but is inverted in IVR.*

II as well (Fig. 82). Involvement of the adjacent interventricular septum gives rise to prominence of the Q wave producing a Q_1T_1 pattern. The T wave is also inverted in CR_4 or IVR and to a less extent as a rule in CR_7. In time the deformed T wave may recover in lead II, but it is seldom wholly corrected in lead I and still less frequently in CR_4. Similarly, changes are usually obvious in CR_1 when they are slight (Fig. 83) or absent (Fig. 84) in lead I.

Posterior infarction.—The changes conform to the T_3 pattern of Parkinson and Bedford where the T wave is inverted in lead III and in lead II (Fig. 85). A prominent Q wave is a commoner feature of posterior than anterior infarction, and is seen in leads III and II, forming a Q_3T_3 pattern. The T wave is not expected to be inverted in CR_4, but as a rule it is either low or inverted in CR_7. In time

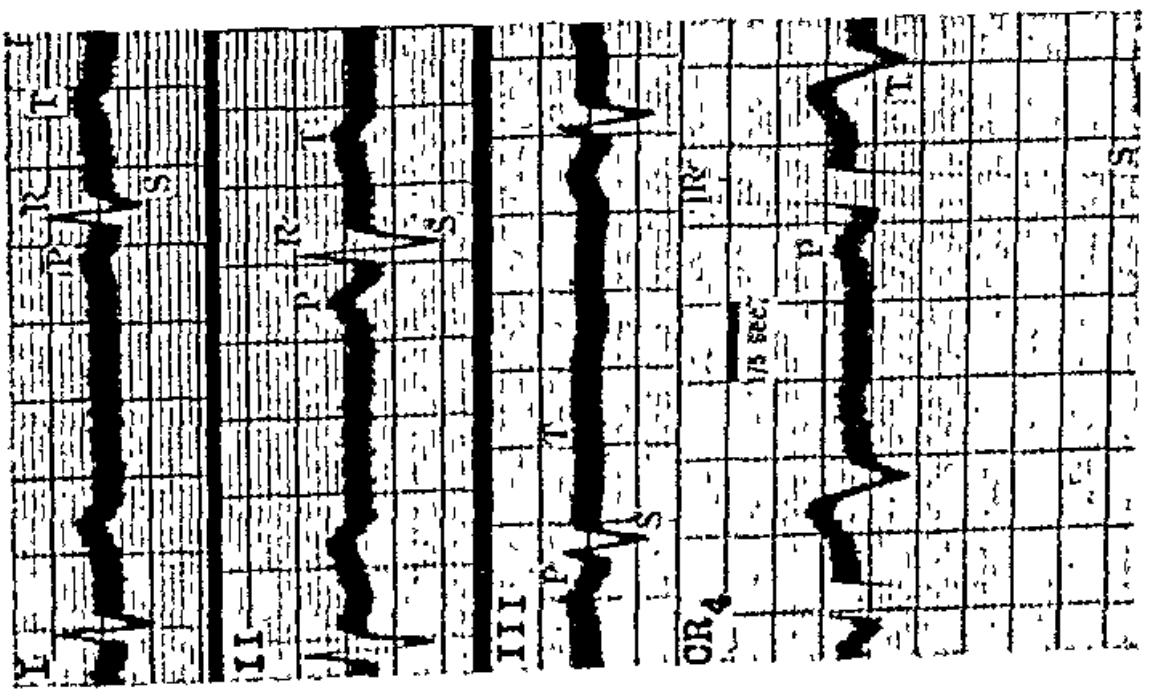


FIG. 84.—The rate is normal. The rhythm is regular. The P-R period is normal. No axis deviation. The T wave is upright in lead I, but is inverted in CR_4 with elevation of the R-T segment.

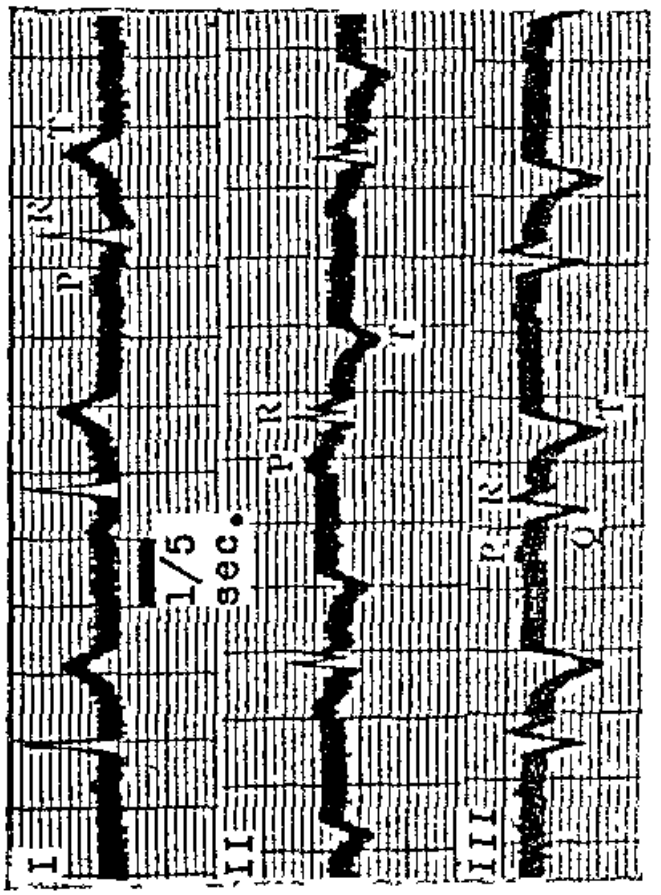


FIG. 85.—The rate is normal. The rhythm is regular. The P-R period is normal. L.A.D. The Q wave is deep in lead III and the T wave is inverted in leads II and III.

the deformed T wave may recover in CR_7, and it may reach the isoelectric level or higher in II, but it never recovers in lead III where the inverted T wave is not corrected by deep inspiration.

Combined anterior and posterior cardiac infarction will cause inversion of the T wave in all three limb leads (Fig. 86) so that concordancy applied to the inverted T waves, cannot then indicate pericarditis in cardiac infarction, for it is not analogous with such relationship between deviation of the R–T segment in leads I and III which exists when acute pericarditis is found by itself.

Infarction, mainly of the *septum*, or confined to the *left side of the heart*, or *anteriorly and towards the apex*, will produce other combinations of T wave inversion ; thus the T wave may be inverted in I and in CR_7 but upright in CR_1.

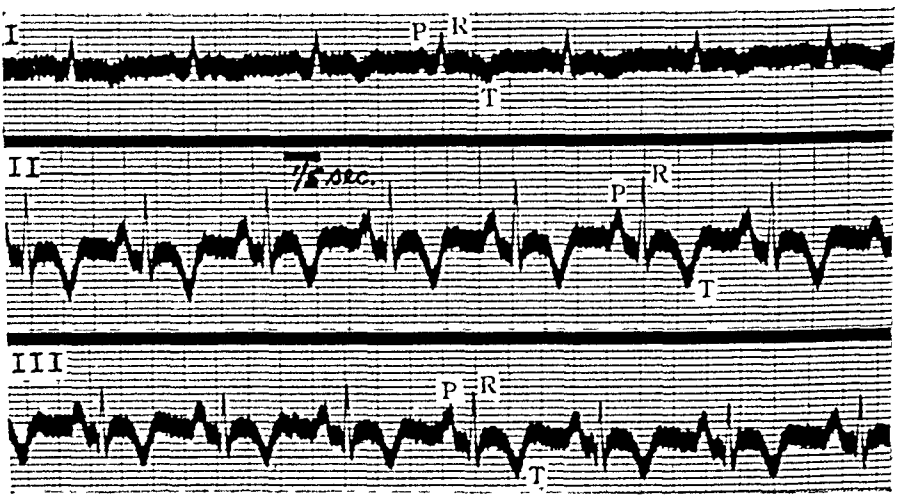


FIG. 86.—The rate is normal. The rhythm is regular. The P–R period is normal. No axis deviation. The T wave is inverted in leads I, II. and III.

CARDIAC ISCHAEMIA

In only a small percentage of cases does the cardiogram change during the attack of cardiac ischaemia. The change takes place in the R–T segment and in the T wave as in cardiac infarction but it is short-lived and lasts less than an hour.

It is common, however, to find the typical cardiographic changes identified with cardiac infarction, in a patient who gives a history of only brief paroxysms of pain on exertion (cardiac ischaemia or angina pectoris of effort). It is likely that such changes are the outcome of a small area of infarction and not of ischaemia only. Certainly the clinical history differs in the two groups of cases although the cardiogram can be identical.

Before leaving a discussion of the relation of cardiographic changes to coronary disease, an answer should be attempted to certain pertinent questions.

First, in the presence of cardiac infarction can the electrocardiogram be normal ?

Although a normal tracing may be found in the presence of severe coronary atheroma it is unlikely that a normal cardiogram is ever obtained when infarction has taken place and especially when chest leads are included

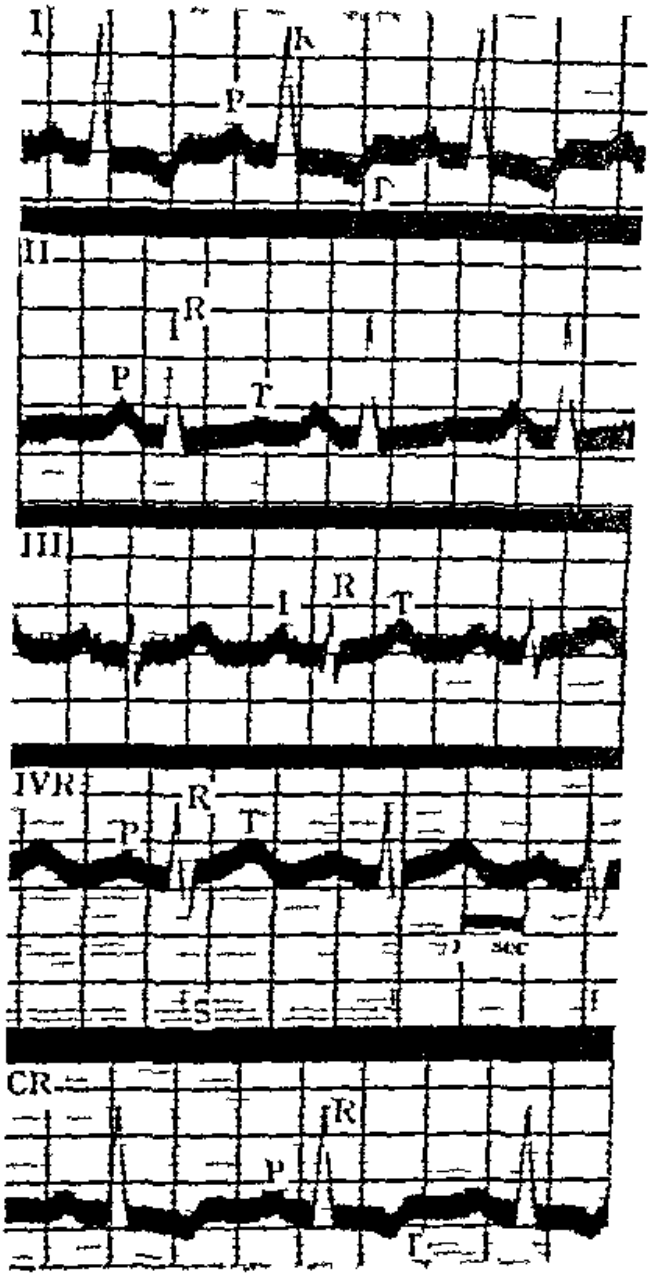


FIG 87.—The rate is normal. The rhythm is regular. The P R period is normal. No axis deviation. The T wave is low in II and is inverted with R T depression in leads I and CR.

Secondly, when the abnormal cardiogram identified with cardiac infarction is fully developed does it ever recover? It is likely that it does not entirely revert to the normal in the great majority of cases especially if the chest lead is included in this analysis but it is true that many of the changes become modified

Thirdly, can the cardiographic changes of cardiac infarction take place in the absence of pain? When such circumstances appear to be present it is usual that the patient admits to tightness in the chest, or it may have been over-

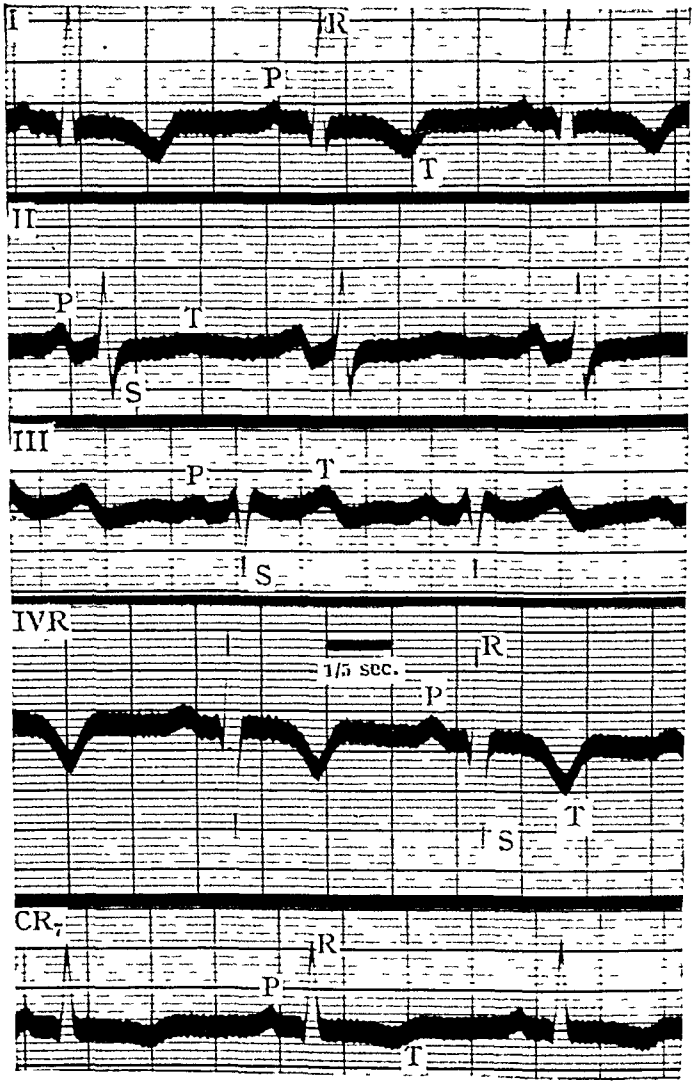


FIG. 88.—The rate is normal. The rhythm is regular. The P–R period is normal. L.A.D. The T wave is flat in lead II and inverted in I, IVR and CR_7; the inversion in IVR is greater than in CR_7.

shadowed by breathlessness from accompanying heart failure, or the patient is an unreliable witness of his symptoms, and makes light of his complaint when he has been rid of it.

HYPERTENSION

When cardiac enlargement is not an obvious finding in a patient with hypertension the electrocardiogram may be normal or at the most it may only show left

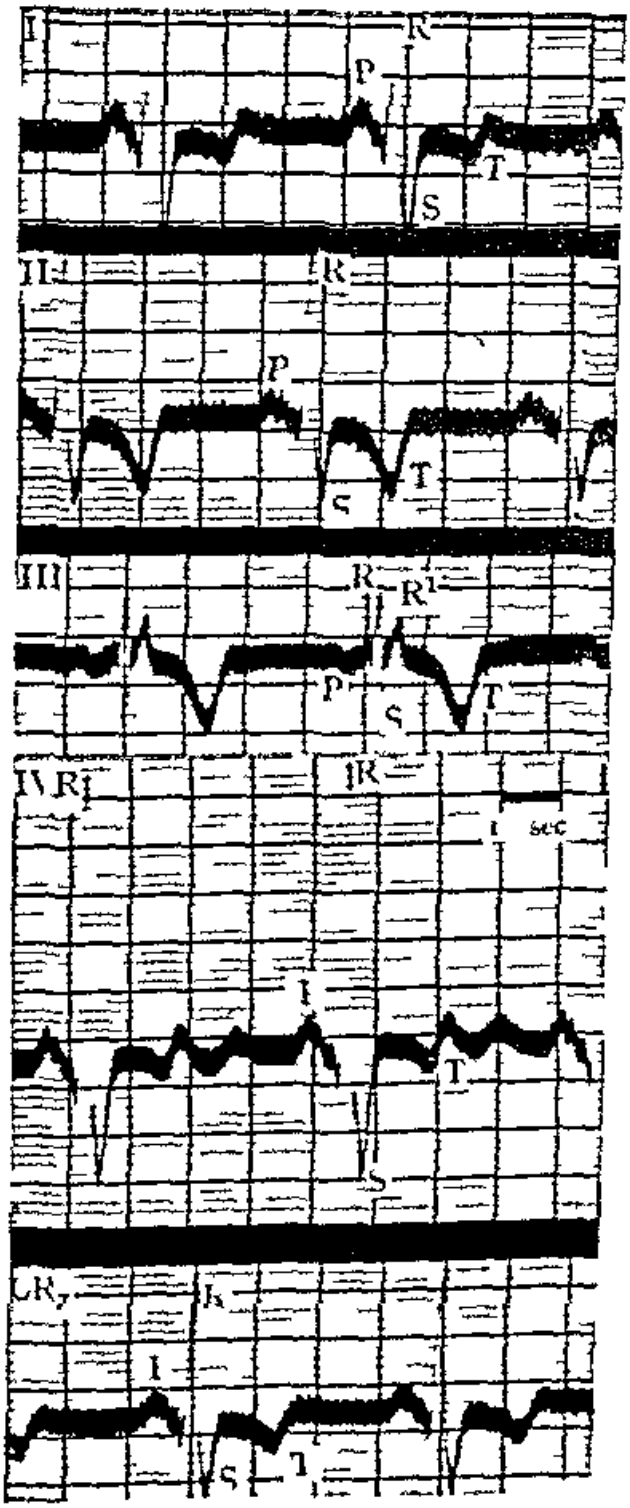
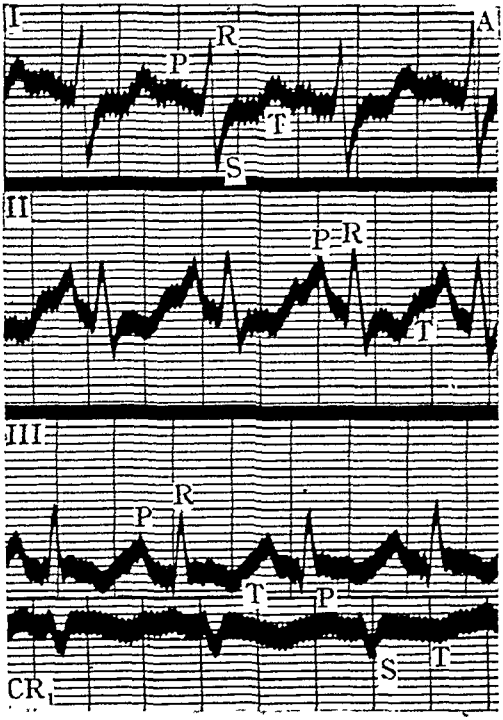
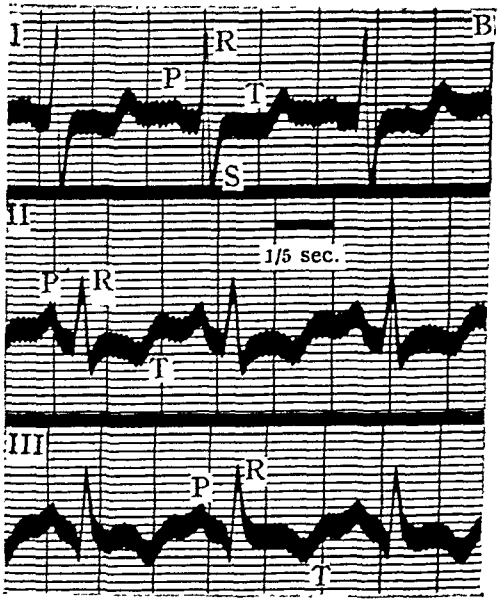


Fig. 89.—The rate is normal. The rhythm is regular. The P-R period is normal. No axis deviation. The T wave is diphasic in leads I and IVR and inverted in II, III and CR.





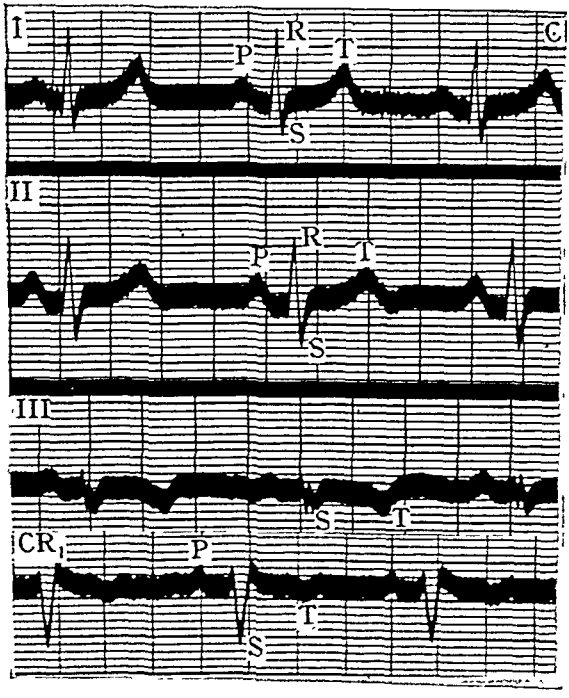


FIG. 90.—The rate is rapid in (A). The rhythm is regular. (A) shows the changes of right bundle branch block. (B) with inverted T waves in leads II and III shows right heart preponderance. The curve has mostly recovered in (C) except for inversion of the T wave in leads III and CR_1.

axis deviation When enlargement of the heart is present or prominent the cardiographic signs of left ventricular preponderance are common These consist of inversion of the T wave in leads I and CR_7 should it be inverted in IVR as well this is less than in CR_7 The R–T segment is depressed Left bundle branch block is also a common curve in hypertension with great cardiac enlargement

When attacks of cardiac ischaemia are a feature of a patient with hypertension the diagnosis of cardiac infarction as a complication is sometimes difficult on

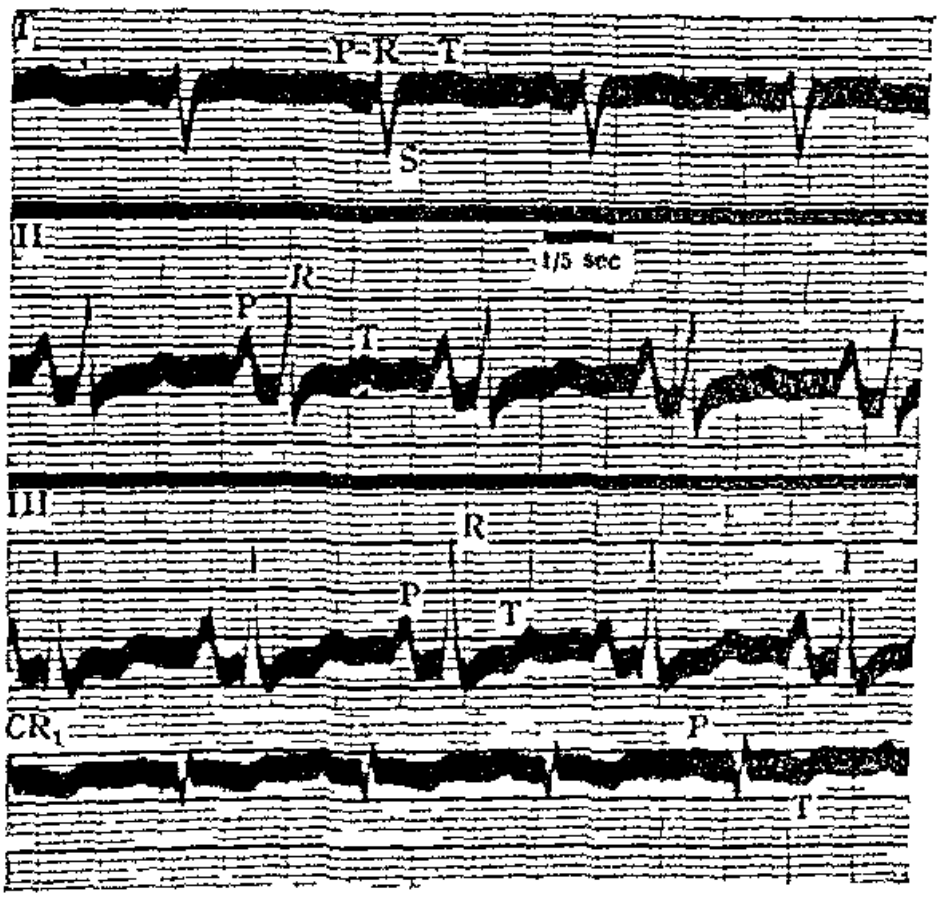


FIG 91 —The rate is normal The rhythm is regular The P R period is normal R A D The P wave is tall in leads II and III The T wave is low in I d phasic in II and III and inverted in CR

cardiographic grounds If the T wave is inverted in leads I and CR and is upright in IVR it is likely that cardiac infarction is absent (Fig 87) although it would be more likely if hypertension was present without pain but should the T wave in IVR be inverted the degree of inversion will help to decide Thus if infarction of the anterior part of the heart has been added to hypertension the inversion is greater in IVR than in CR (Fig 88) whereas the reverse is true of uncomplicated hypertension (Fig 5) or of hypertension with posterior cardiac infarction or aortic valvular disease (Fig 89)

LUNG DISEASE

Pulmonary embolism

Sudden extensive infarction of the lungs from embolism produces a right bundle branch block in the electrocardiogram. If the patient survives, changes characteristic of right ventricular hypertrophy take place, namely, inversion of the T wave in leads II, III and CR_1, along with right axis deviation (Fig. 90). During

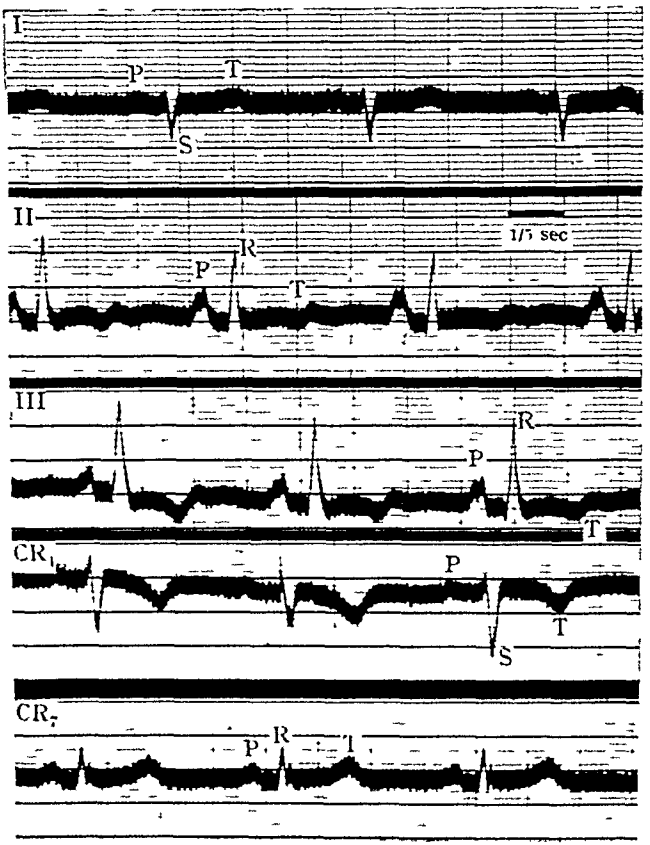


FIG. 92.—The rate is normal. The rhythm is regular. The P–R period is normal. R.A.D. The P wave is tall and broad in leads II and III. The T wave is slightly inverted in II, and more deeply in leads III and CR_1; the T wave is upright in CR_7.

recovery the inverted T in CR_1 is the last to be corrected. The Q wave may be deep in III, and the P wave tall in II and III.

Emphysema

The electrocardiogram in emphysema may be normal, but in the presence of right heart enlargement the tracing may show such abnormalities as a deep S in lead I and inversion of the T wave in CR_1 (Fig. 91). If there is much enlargement the

T wave is also inverted in leads II and III (Fig 92) as an expression of conspicuous right ventricular preponderance

Primary pulmonary hypertension

This condition may resemble pericardial disease in some of its clinical and radiological manifestations but its cardiographic pattern serves to distinguish

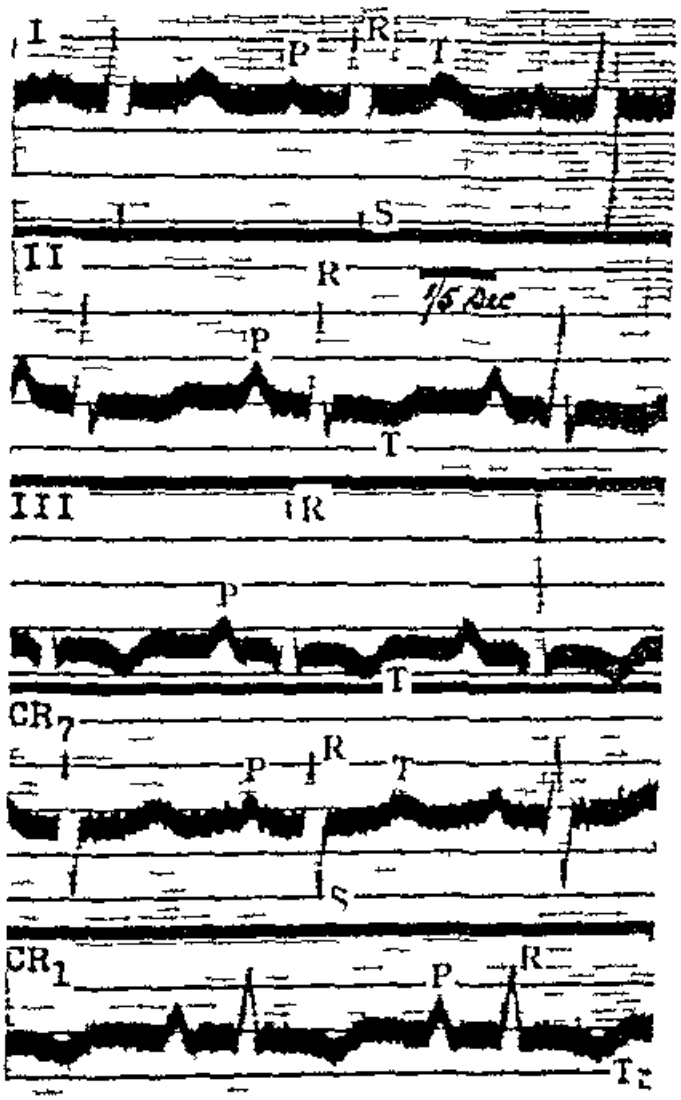


FIG 93.—The rate is normal. The rhythm is regular. The P R period is slightly prolonged. R A D. The T wave is inverted in leads II, III and CR_1 but upright in CR

it, for there is right axis deviation in addition to inversion of the T waves in leads II, III and CR_1 (Fig 93)

Pneumonectomy and pneumothorax

It is not unusual for auricular fibrillation or auricular tachycardia to set in after pneumonectomy but the sudden depletion of so large a part of the pulmonary

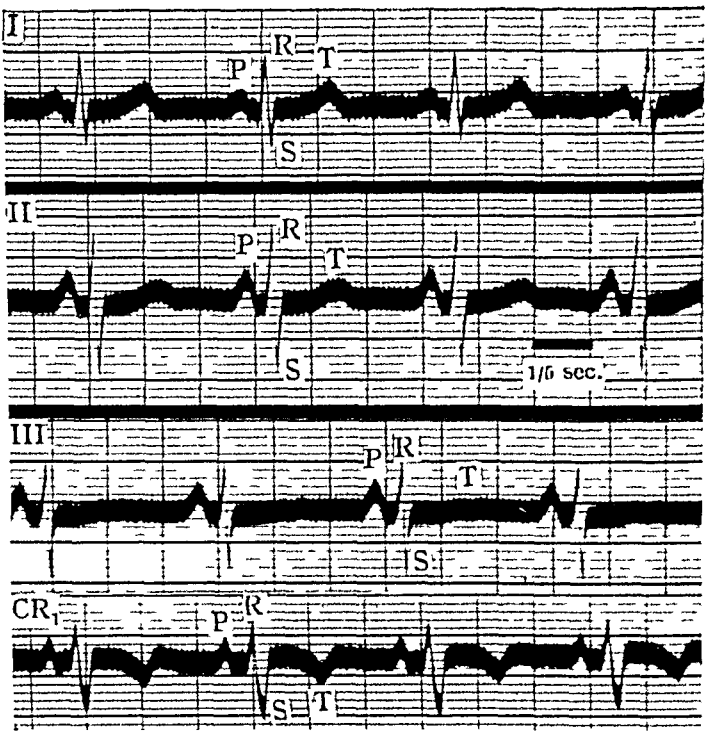


FIG. 94.—The rate is a little rapid. The rhythm is regular. The P–R period is rather short. No axis deviation. The T wave is flat in lead III and inverted in CR_1.

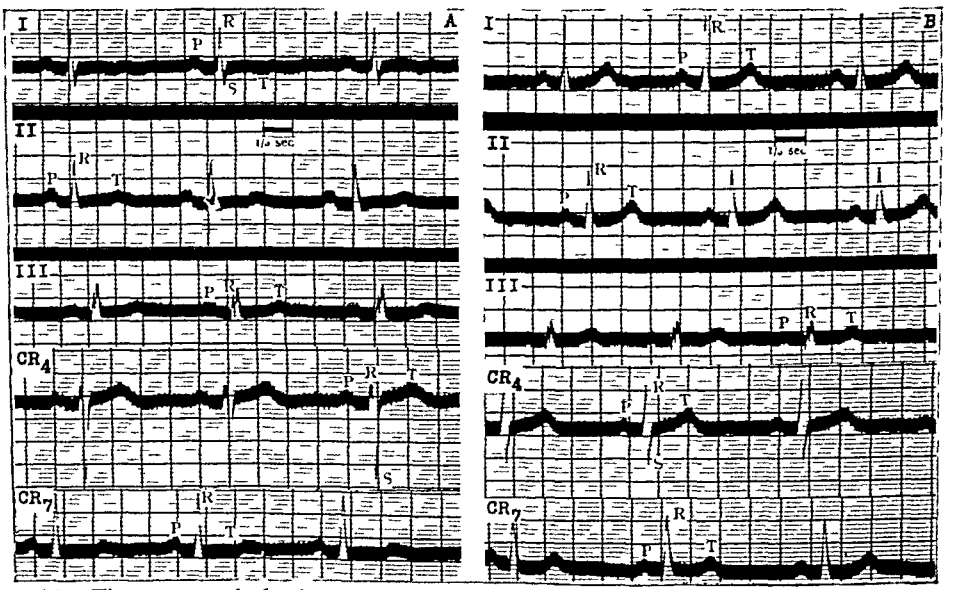


FIG 95.—The rate is a little slow. The rhythm is regular. The P–R period is full No axis deviation In (A) the T wave is inverted in lead I, upright in CR_4, and low in II and CR_7. In (B), taken after three weeks on thyroid treatment, the T waves are upright and normal.

circulation does not give rise to cardiographic changes common to right ventricular preponderance

Minor changes, particularly electrical variation (Fig 57) may take place in the electrocardiogram at the initiation of spontaneous pneumothorax

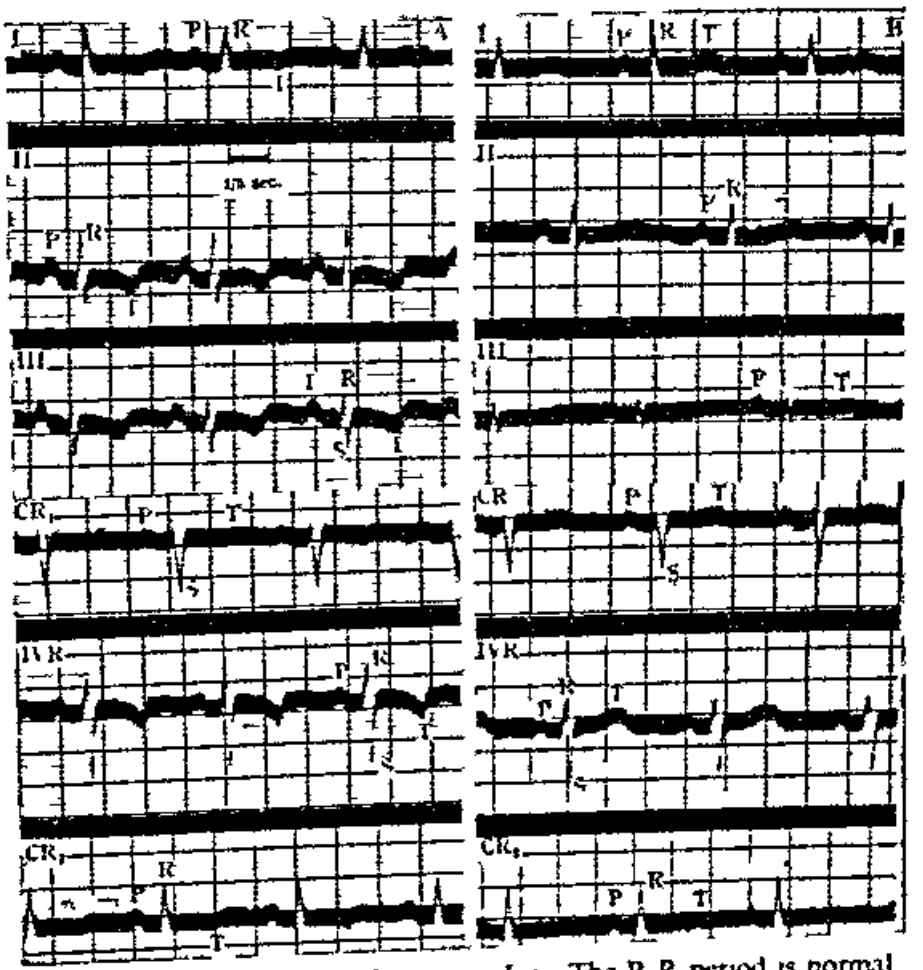


FIG 96.—The rate is normal The rhythm is regular The P R period is normal In (A) the T waves are inverted in most of the leads but they are upright in (B) after treatment with desoxycorticosterone acetate

ENDOCRINE DISORDERS

Thyroid toxaemia

Sinus tachycardia is the commonest cardiographic abnormality in thyroid toxaemia and auricular fibrillation is another common form of arrhythmia Amongst the lesser changes are those which tell of a degree of right ventricular preponderance, namely a low T wave in lead II and inversion of the T wave in leads III and CR_1 (Fig 94) Such cardiographic changes predict enlargement of the right heart from the toxic effects of the goitre, and it is accompanied by triple heart rhythm from the addition of the third heart sound

Myxoedema

The electrocardiogram of myxoedema characteristically shows sinus bradycardia,

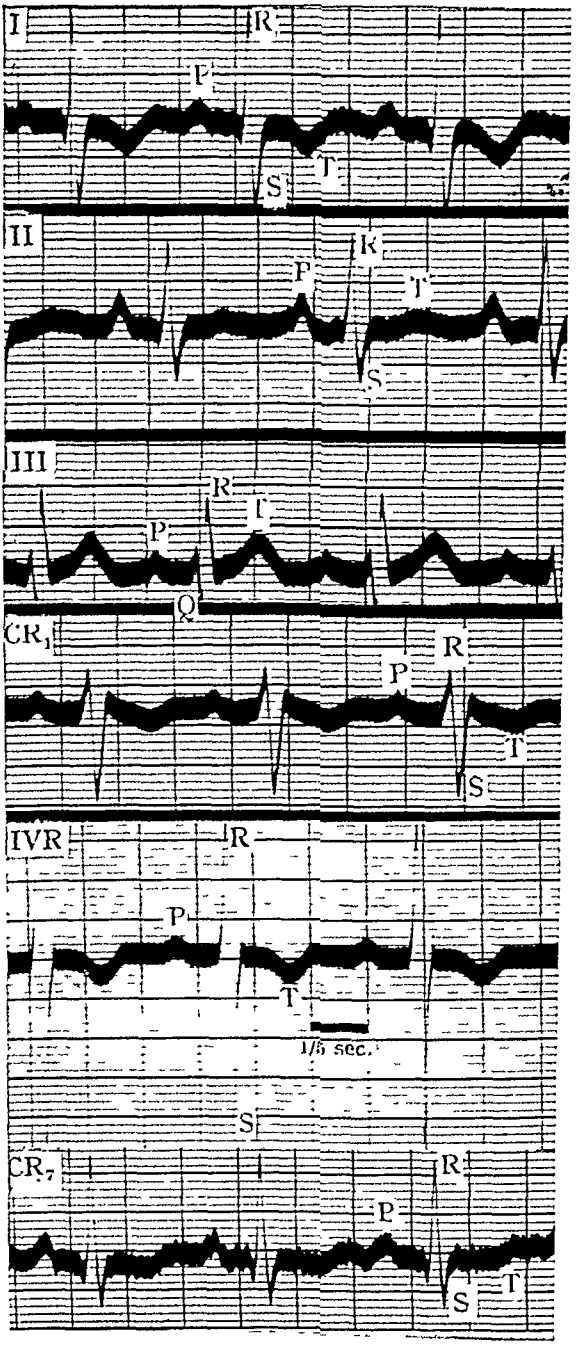


FIG. 97.—The rate is normal. The rhythm is regular. The P–R period is normal. No axis deviation. The T wave is inverted in leads I, CR_1, and IVR ; it is diphasic in CR_7.

and low amplitude of the waves with flattening or inversion of the T waves Such changes are corrected after a short period of thyroid therapy (Fig 95)

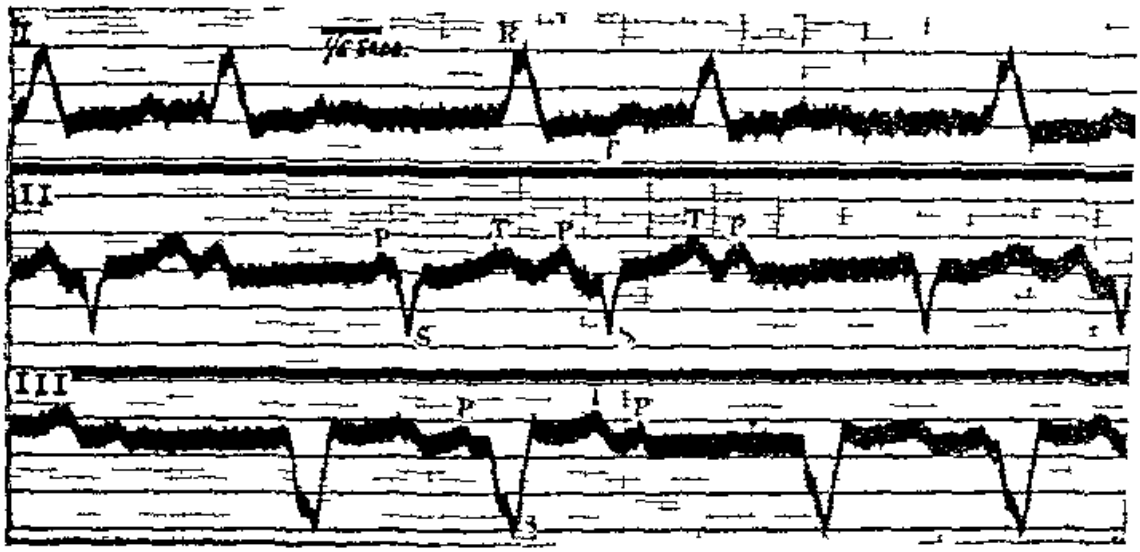


FIG 98 —The rate is slow The rhythm is irregular from complete heart block with alternating ventricular extrasystoles Left bundle branch block

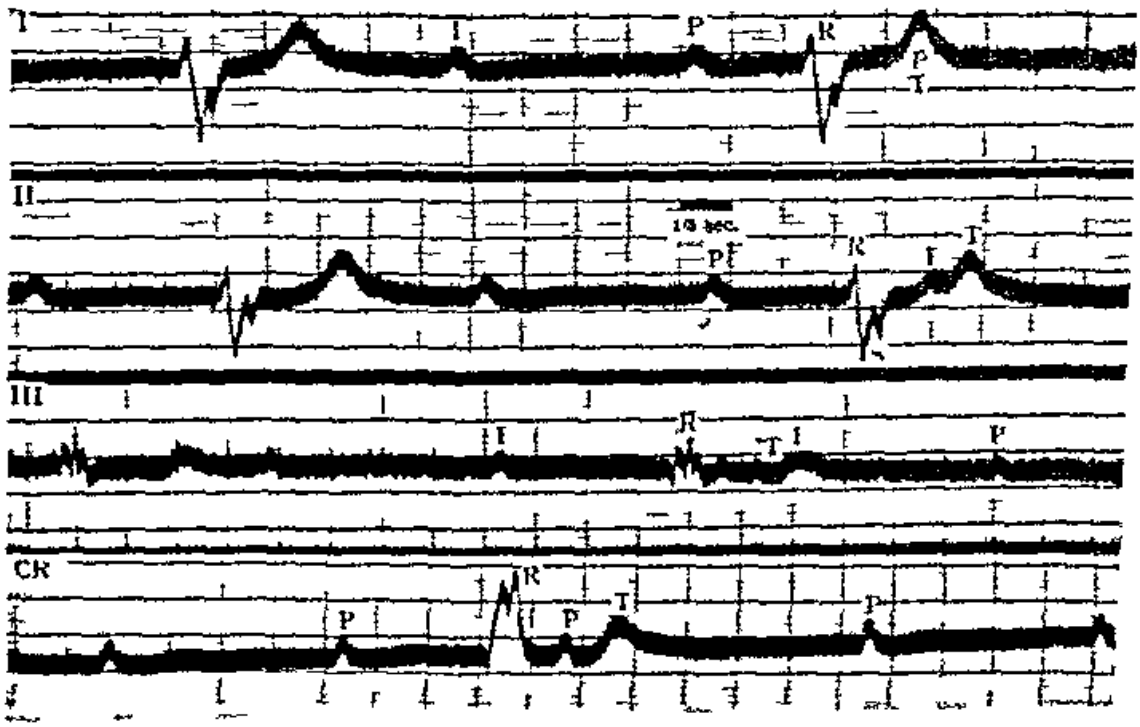


FIG 99 —The rate is very slow and the rhythm is abnormal from complete heart block R A D and right bundle branch block

Addison's disease

Although the electrocardiogram in this condition is usually a normal tracing at times it may show prominent irregularities in the form of inversion of the

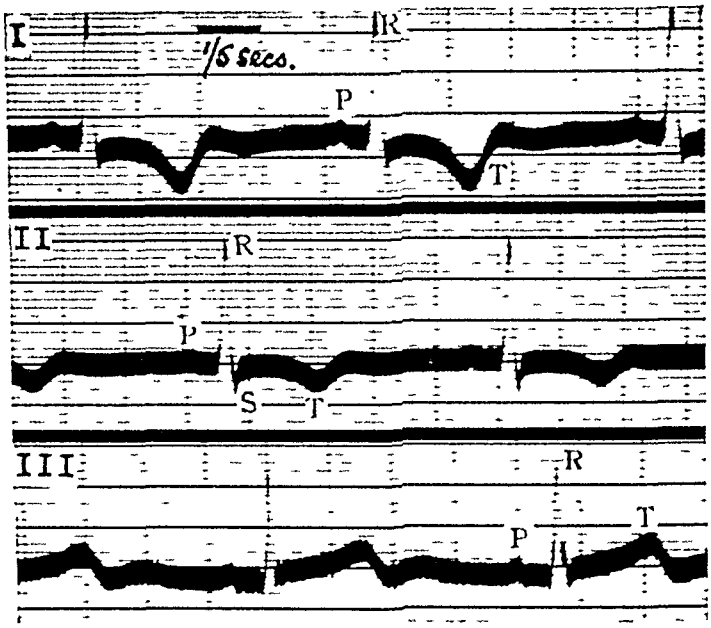


FIG. 100.—The rate is slow. The rhythm is regular. The P–R period is normal. No axis deviation. The P waves are small and the T waves are inverted in leads I and II.

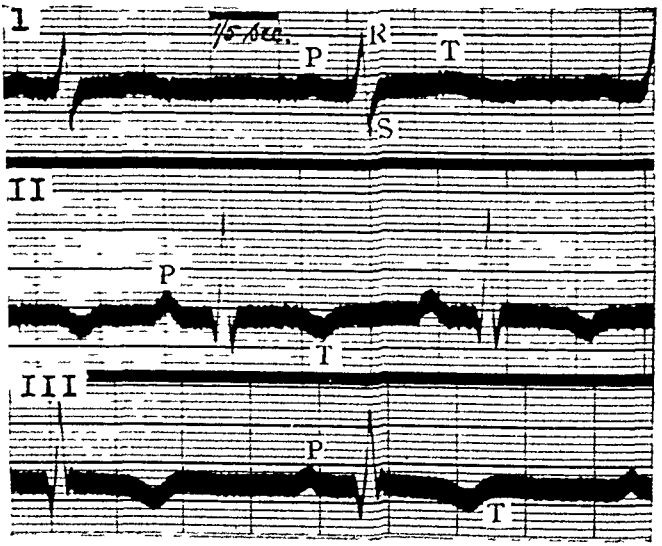


FIG. 101.—The rate is slow. The rhythm is regular. The P–R period is normal. No axis deviation. The T wave is low in lead I and inverted in leads II and III.

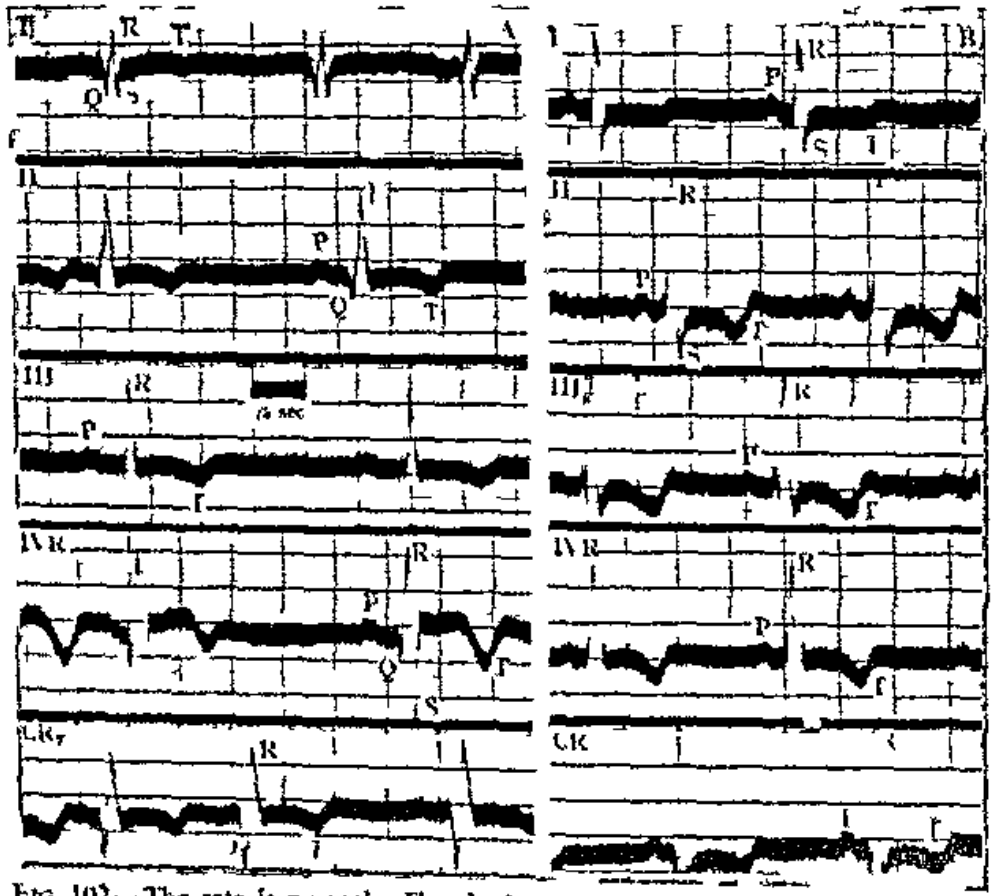


Fig 102.—The rate is normal The rhythm is regular except for extrasystoles in (A) The P R period is normal No axis deviation Inversion of the T wave in all leads in both (A) and (B) cardiograms taken of two patients with Friedreich disease.

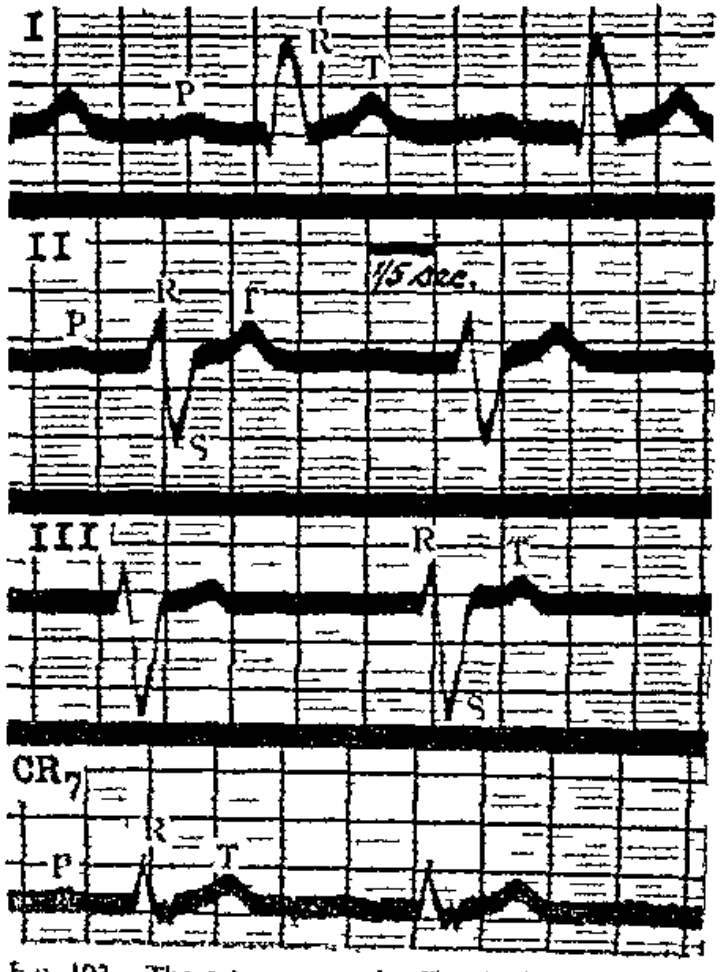


Fig 103 The rate is normal The rhythm is regular The P R period is prolonged L A D The P wave is small The QRS complexes are wide

T waves. Such changes are not constantly related to the serum potassium value, and they are corrected by treatment with desoxycorticosterone acetate (Fig. 96).

Suprarenal tumour

The electrocardiogram is a valuable test when the diagnosis of suprarenal tumour is in doubt. It may show inversion of the T wave in one or more leads (Fig. 97), and this change may take place in the absence of maintained hypertension.

DISORDERS OF THE CENTRAL NERVOUS SYSTEM

Friedreich disease

The electrocardiogram is abnormal in about two-thirds of the patients with Friedreich disease, and in one-third the changes are conspicuous. When

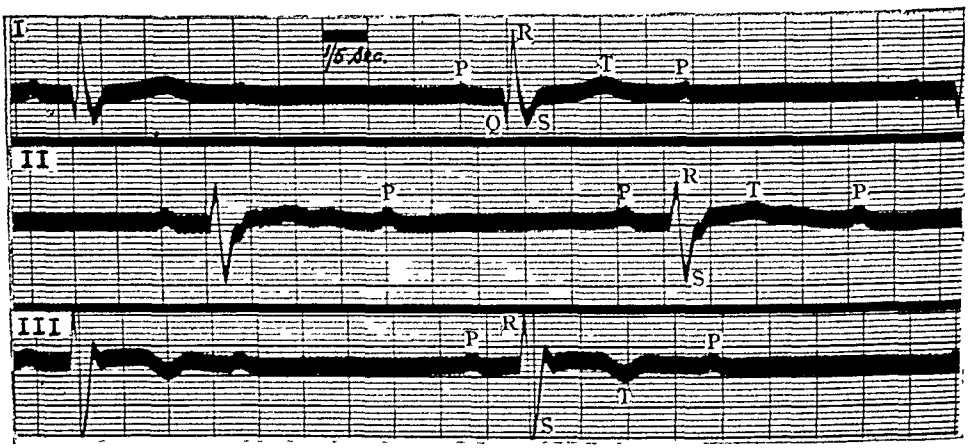


FIG. 104.—The rate is very slow. The rhythm is irregular from 2 to 1 heart block. L.A.D. The T wave is inverted in lead III.

Stokes-Adams disease is added the cardiogram shows complete heart block (Figs. 98 and 99). Other tracings show a coronary type of curve of the T_1 variety (Fig. 100), or the T_3 variety (Fig. 101), while others may show T inversion in all limb leads (Fig. 102).

Myotonia atrophica

The changes which commonly characterize the electrocardiogram of myotonia atrophica include prolongation of the P–R period, low voltage of the P wave, slurring of the QRS complex, and left axis deviation (Figs. 103 and 104).

Periodic paralysis

The electrocardiogram of familial periodic paralysis is normal except during the attacks when the P–R and QRS periods become prolonged and the T waves low or inverted. The changes are related to the serum potassium value which is lowered during the attack. The cardiogram returns to normal at the end of the paralytic attack, whether spontaneous or brought about by the administration of potassium salts.

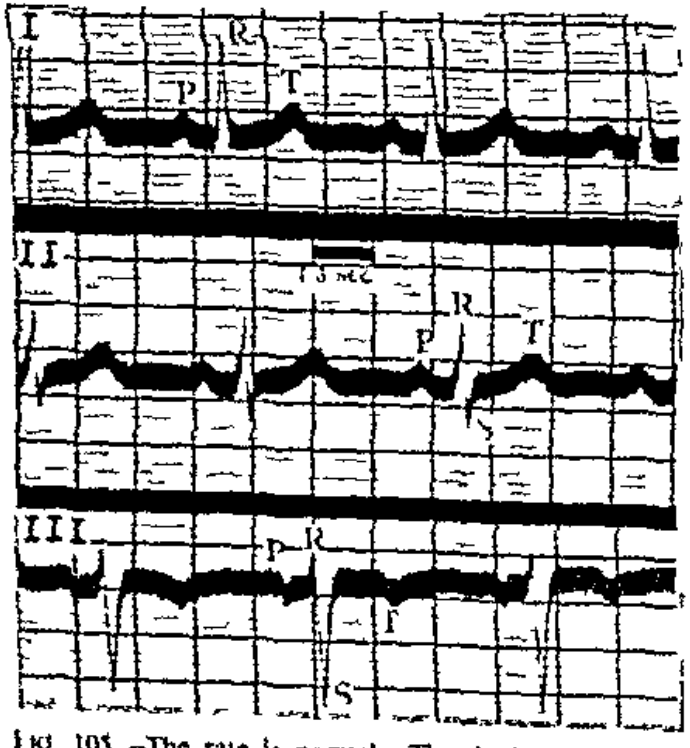


FIG 105 —The rate is normal The rhythm is regular The P R period is normal L A D The P, QRS, and T waves in lead III are inverted

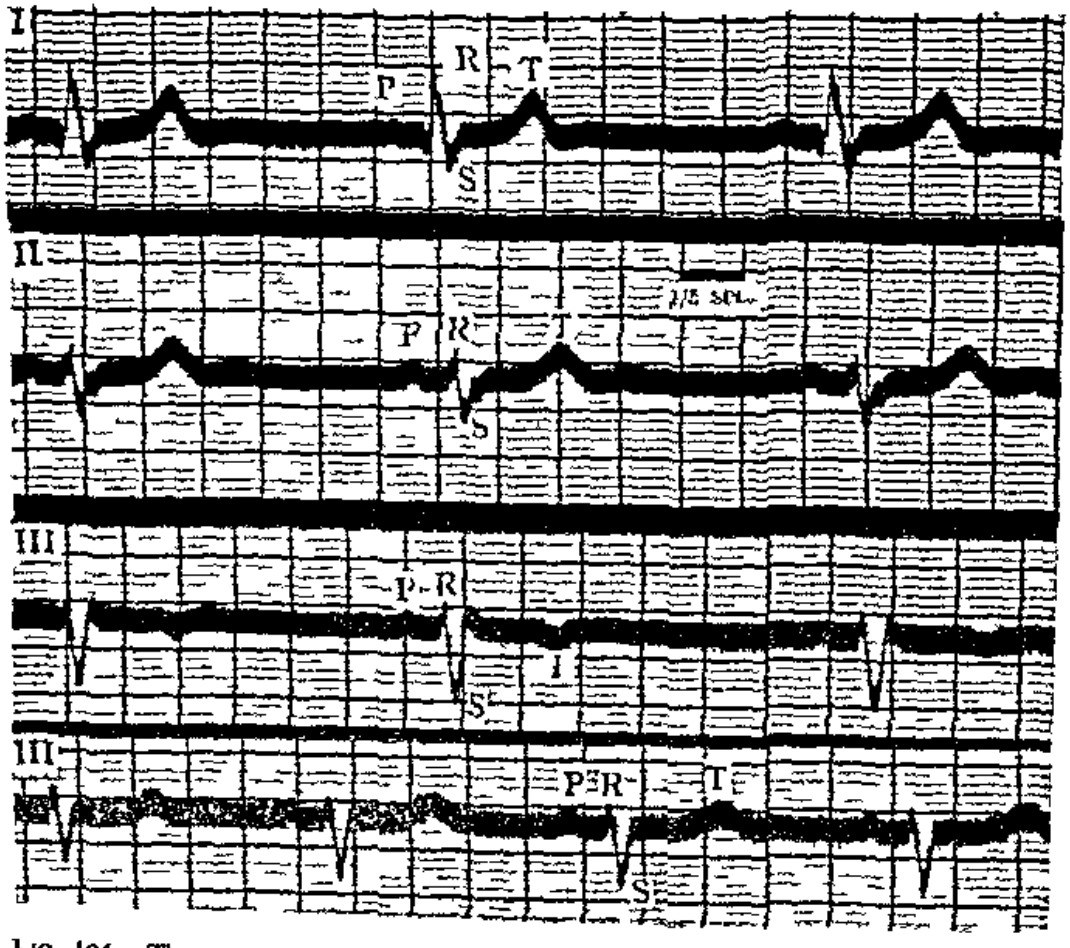


FIG 106 —The rate is normal The rhythm is regular The P R period is normal No axis deviation The inverted T wave in the first lead III is raised in the second lead III recorded during deep inspiration

OTHER CONDITIONS

Diphtheria

The cardiogram is often abnormal in diphtheria and especially when the illness is judged to be severe on clinical grounds. The changes include heart block,

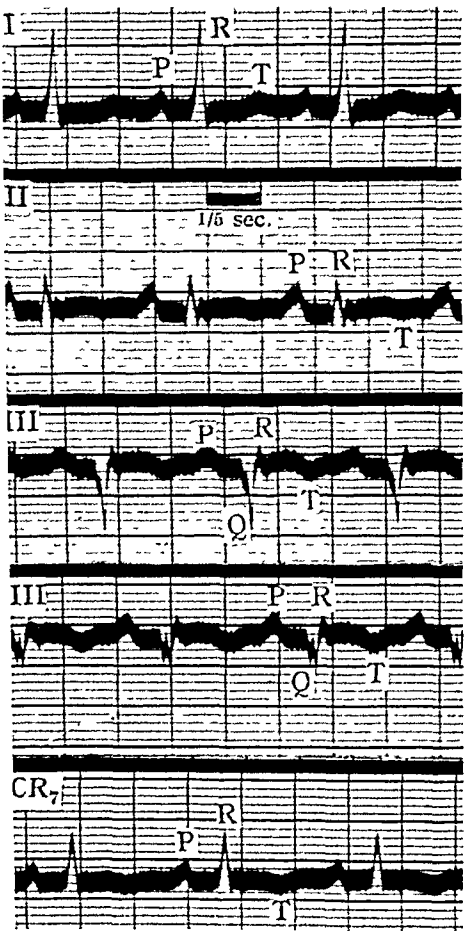


FIG. 107.—The rate is normal. The rhythm is regular. The P–R period is normal. L.A.D. The T wave is low in lead II and inverted in leads III and CR_7. Both the R–T deviation and T wave inversion in lead III are accentuated in the second lead III which was recorded during deep breathing.

bundle branch block, depression of the R–T segment, inversion of the T waves, sinus bradycardia, auricular fibrillation and auricular tachycardia.

POSTURE

Sometimes a low amplitude or even inversion of the T wave in lead II may occur in the sitting or standing posture in health, and in this event recumbency with elevation of the diaphragm at full expiration may correct the deformity.

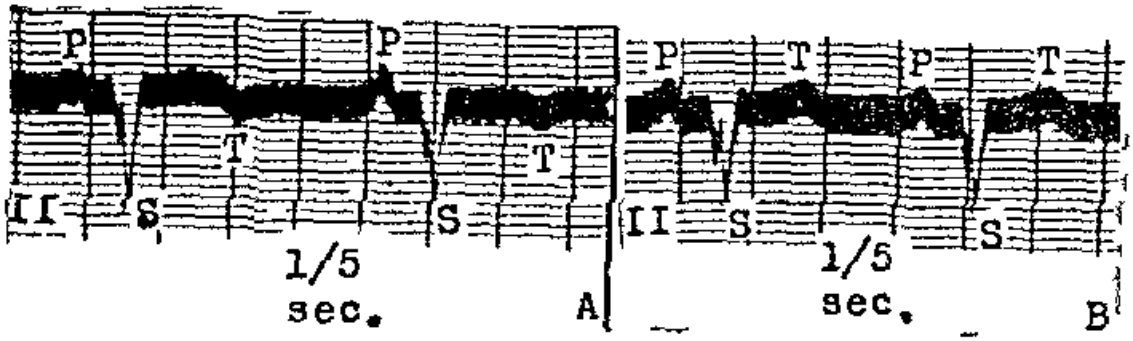


FIG 108.—Inversion of the T wave in cardiac infarction shown in (A) is temporarily raised by inhalation of amyl nitrite as shown in (B)

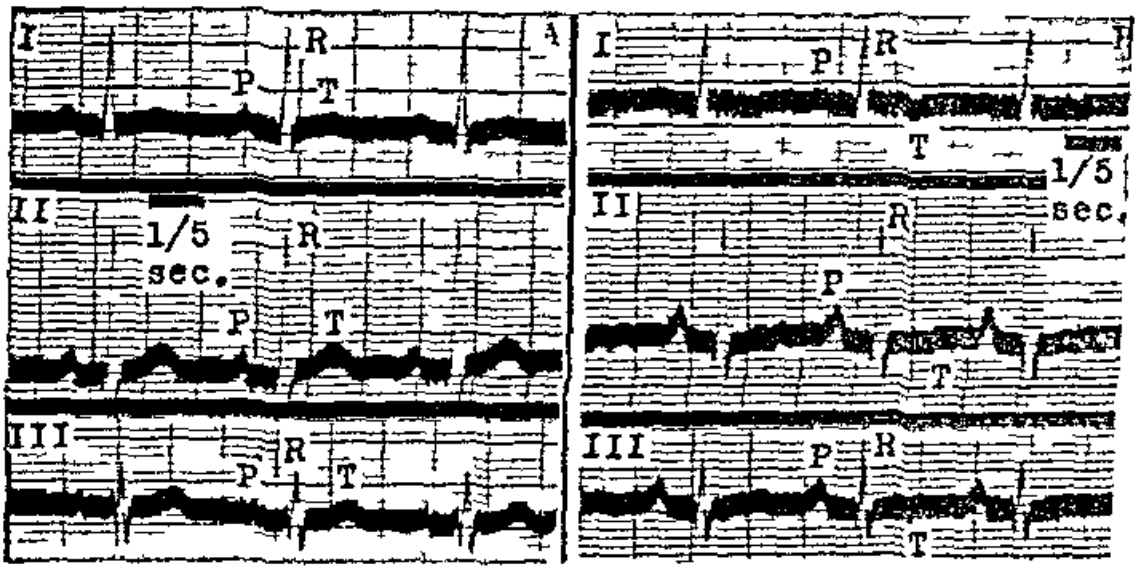


FIG 109.—The rate is normal. The rhythm is regular. The P R period is normal. No axis deviation. The T wave is upright in all leads in (A) but is low or inverted in (B) after digitalisation

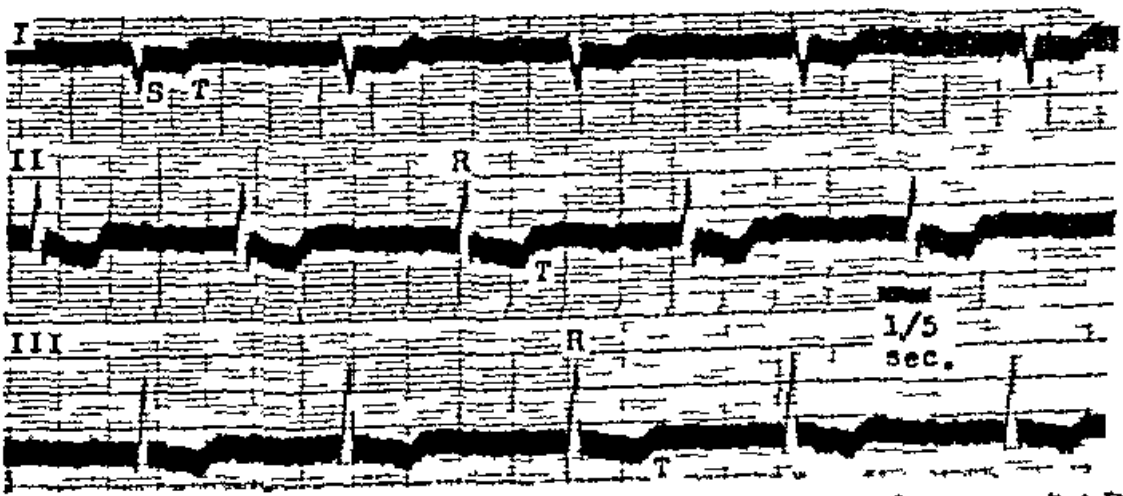


FIG 110.—The rate is normal. The rhythm is irregular from auricular fibrillation. R A D. Small voltage in lead I. The R T segments are depressed in all three limb leads

Of far greater frequency, as the result of elevation of the diaphragm from any cause, are changes in lead III ; these include inversion of the QRS, T and P waves separately or together (Fig. 105). On deep inspiration, which depresses the diaphragm, the deformity is partially or wholly corrected (Fig. 106). Such a test is useful in differentiating these changes from those found in coronary disease or posterior cardiac infarction where the deformed T wave remains uncorrected by deep inspiration (Fig. 107) ; examination of the T wave in leads II and CR_7 will also help in diagnosis.

AMYL NITRITE AND EXERCISE

Inhalation of amyl nitrite will often correct temporarily the inverted T wave found in coronary disease (Fig. 108). This effect is also seen in the case of the deformed T wave of hypertension or aortic valvular disease.

Tachycardia induced by exercise may increase the partly inverted T wave of cardiac infarction, but it may often correct it.

QUINIDINE

The effect of quinidine in auricular tachycardia and auricular fibrillation is to slow the auricular rate and to quicken the ventricular. Its action in sinus rhythm is to lengthen the P–R and QRS periods, and to decrease the height of the T wave.

DIGITALIS

Digitalis is without much effect upon the heart rate in sinus rhythm, slowing it only a little, but the ventricular rate is effectively slowed in auricular fibrillation. Auricular flutter or auricular tachycardia is often converted into fibrillation by digitalis. A characteristic effect of digitalis therapy is depression of the R–T segment, and a decrease in the amplitude of the T wave which eventually becomes inverted and emerged into the depressed segment (Figs. 109 and 110). It often lengthens the P–R period and occasionally it produces partial heart block. In organic heart disease, digitalis in full therapeutic doses frequently gives rise to extrasystoles and this may result in a bigeminal pulse.

TEST ELECTROCARDIOGRAMS

Now that the electrocardiogram for the separate clinical conditions has been described, and the reader is familiar with the scheme already outlined and which facilitates the interpretation of the tracing, it is opportune to exhibit a number of cardiograms for diagnosis. Each one should be read in accordance with the

scheme and a diagnosis pronounced before consulting the key to such test cardiograms which is found on page 92.

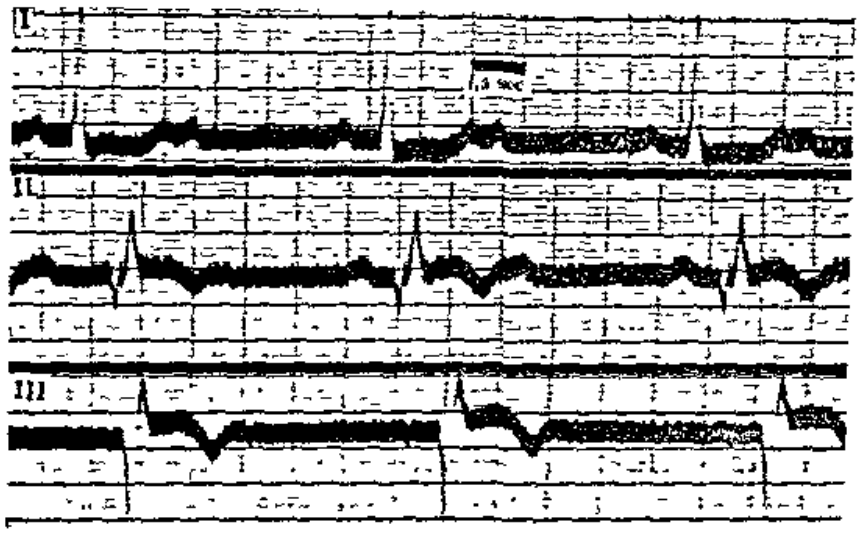


FIG. 111

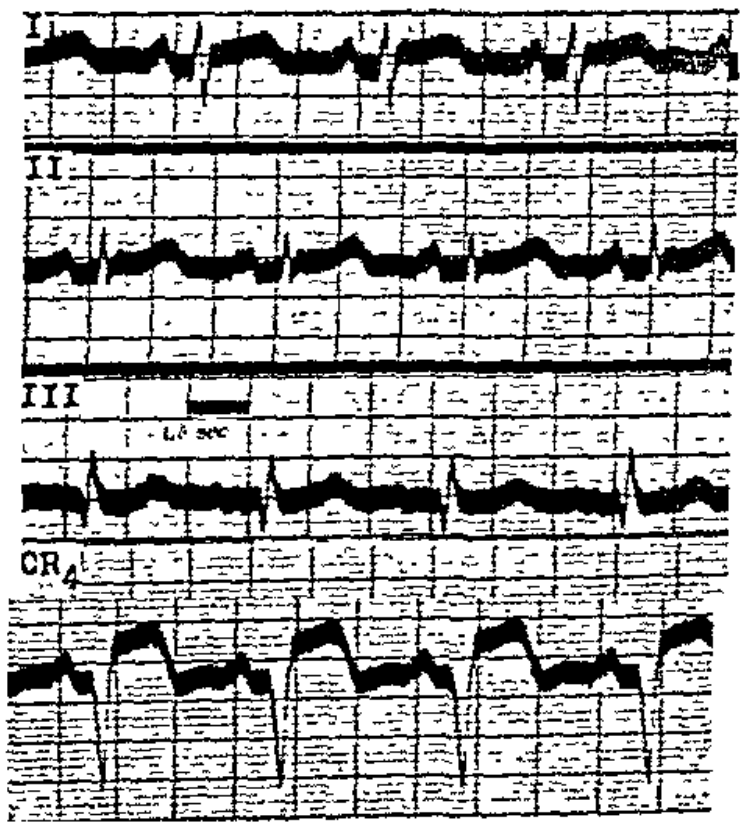


FIG. 112

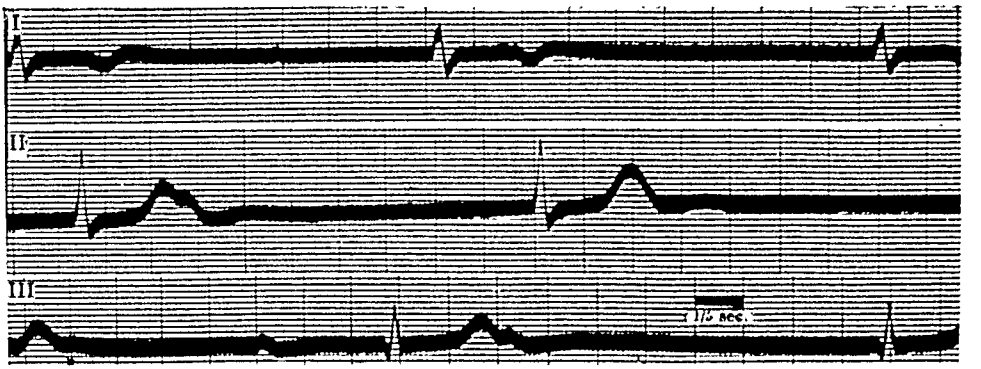


FIG. 113

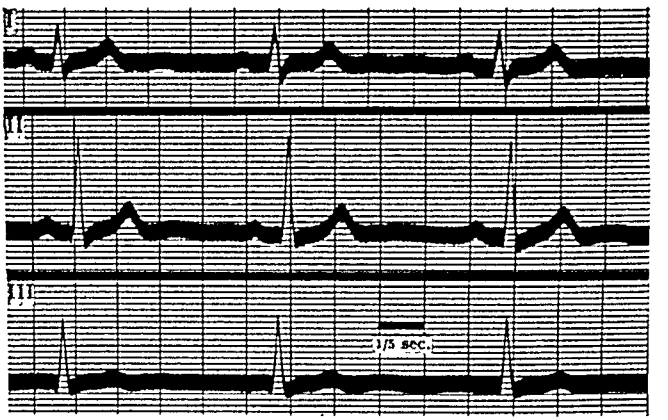


FIG. 114

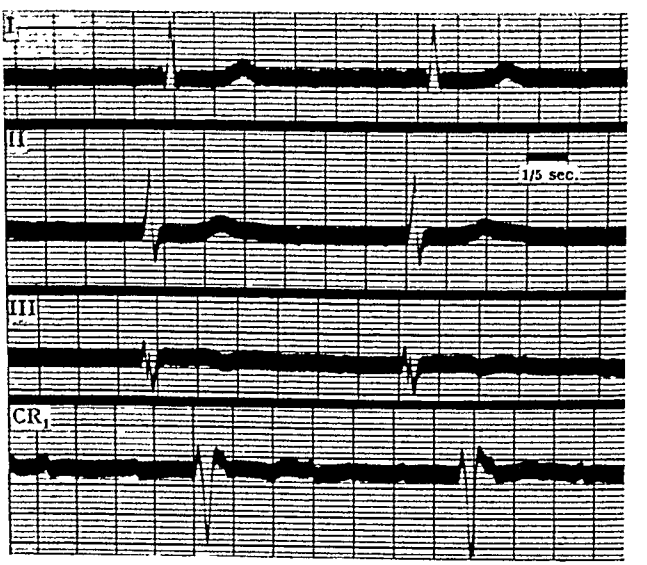


FIG. 115

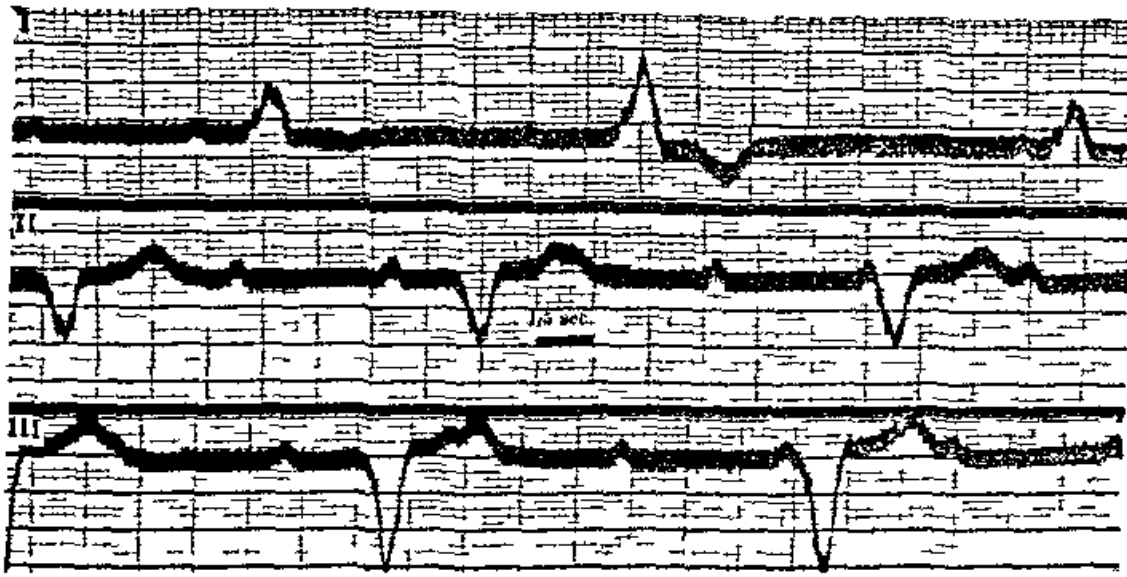


FIG 116

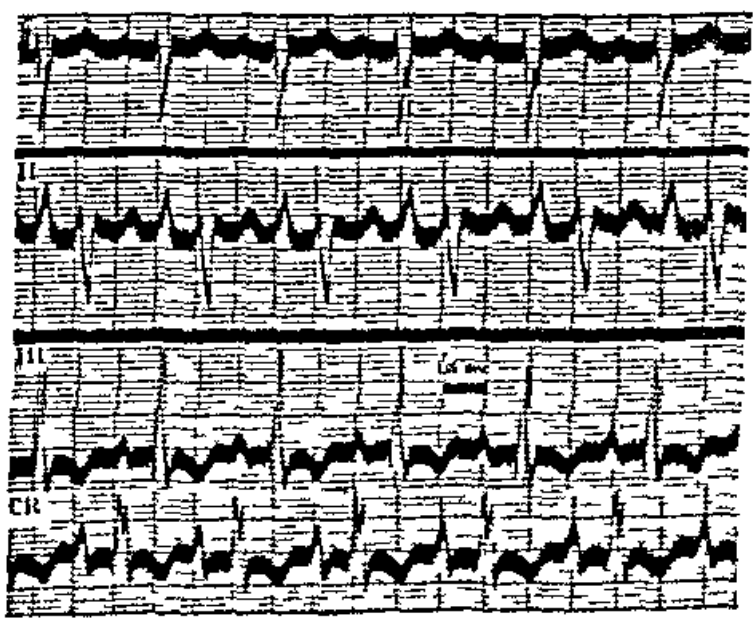


FIG 117

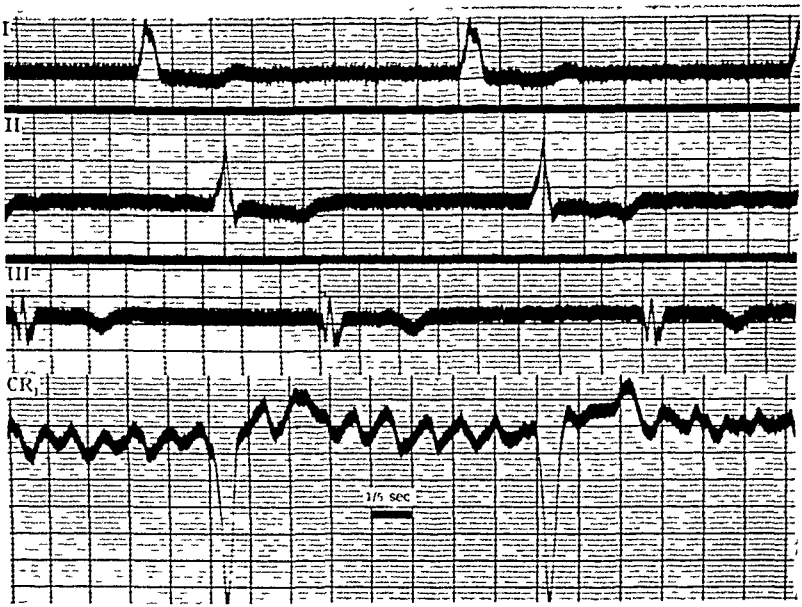


FIG. 118

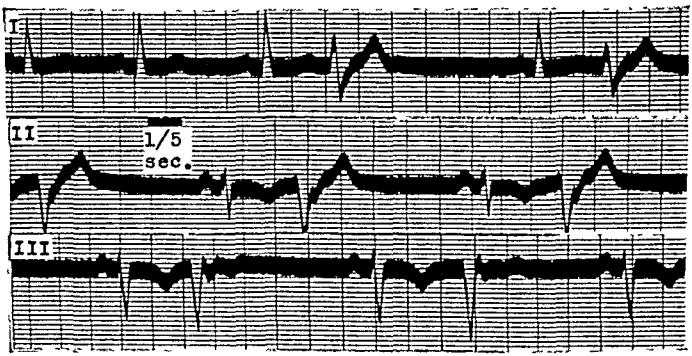
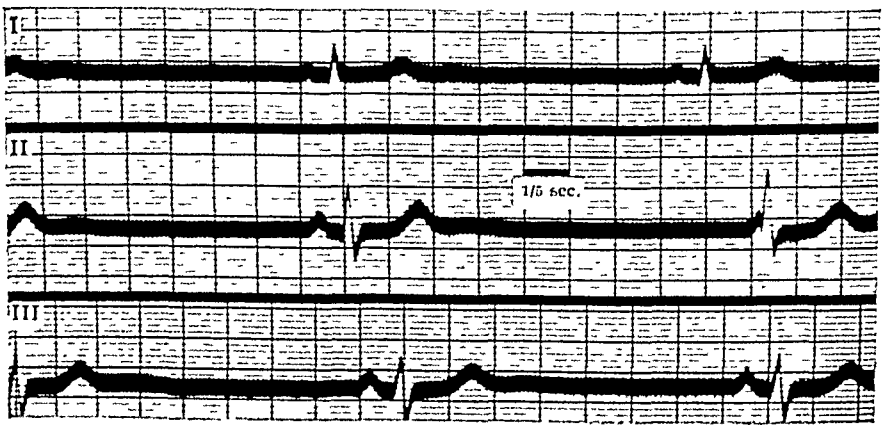


FIG. 119



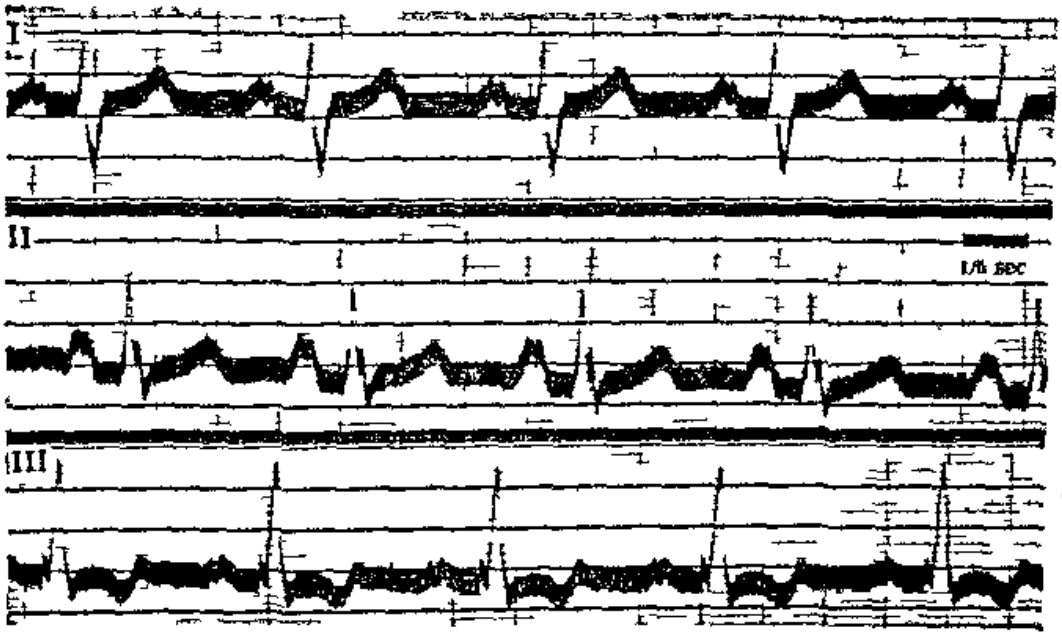


FIG 121

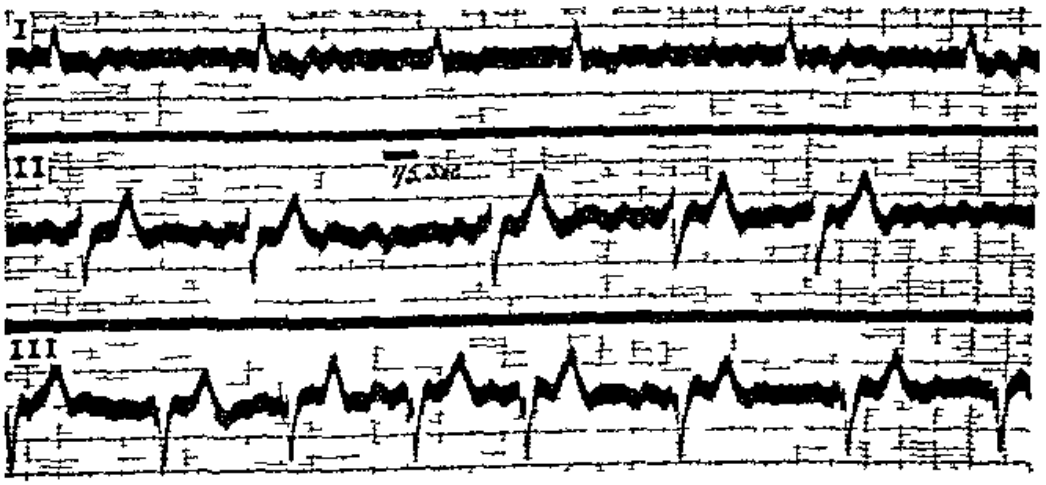


FIG 122

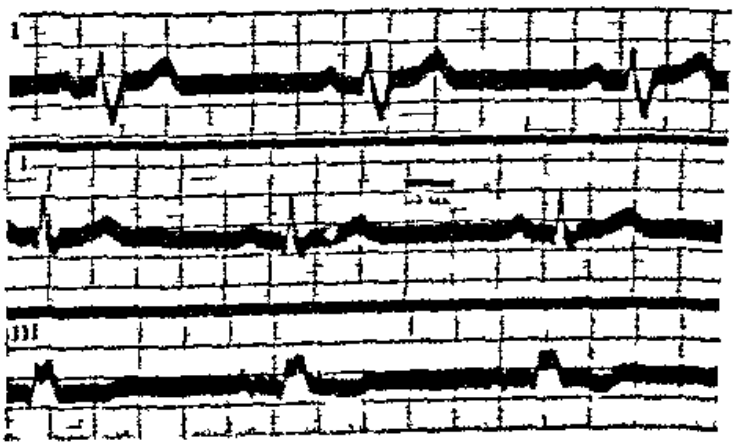


FIG 123

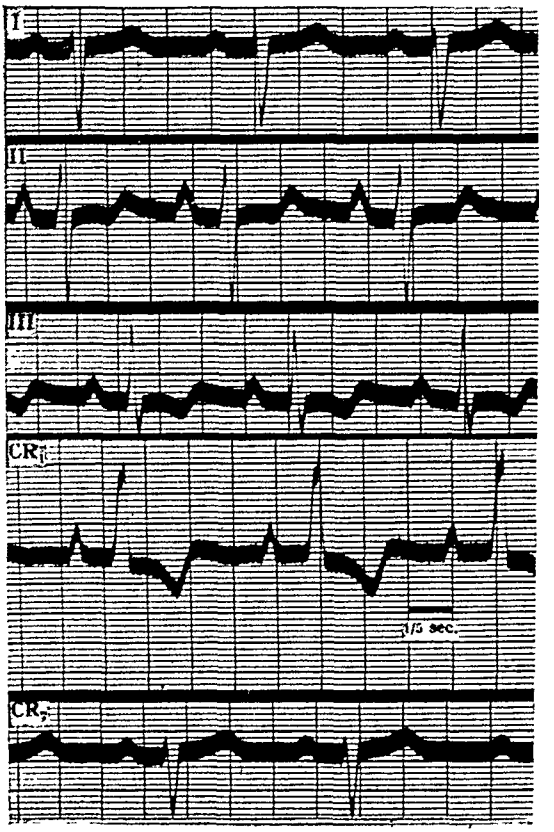


FIG. 124

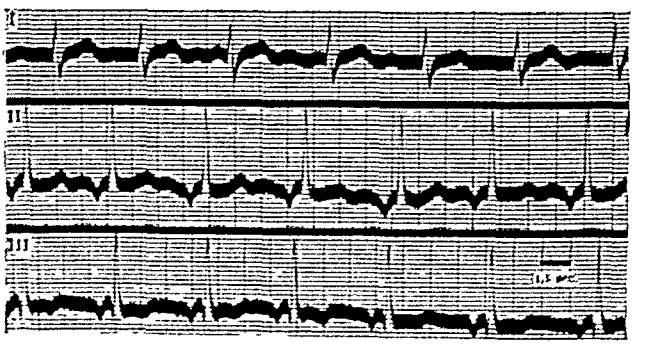


FIG. 125

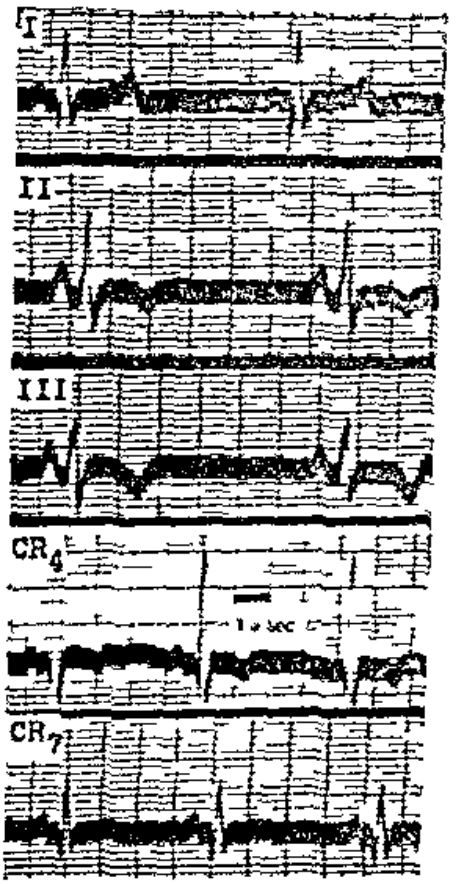


FIG 126

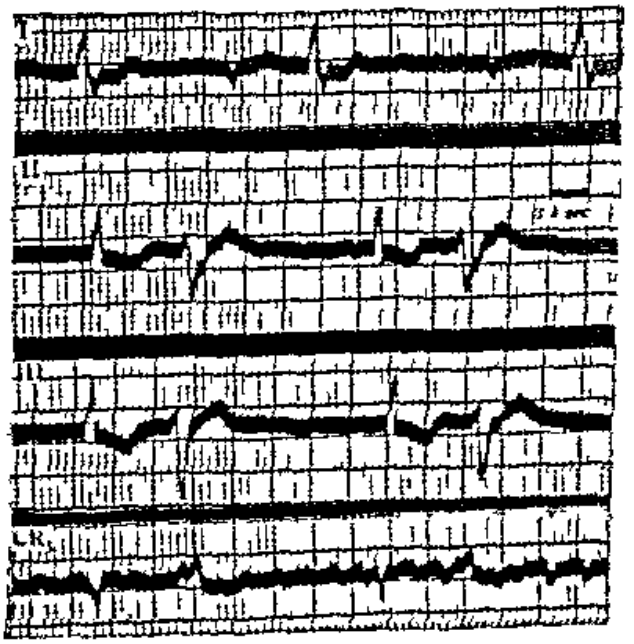


FIG 127

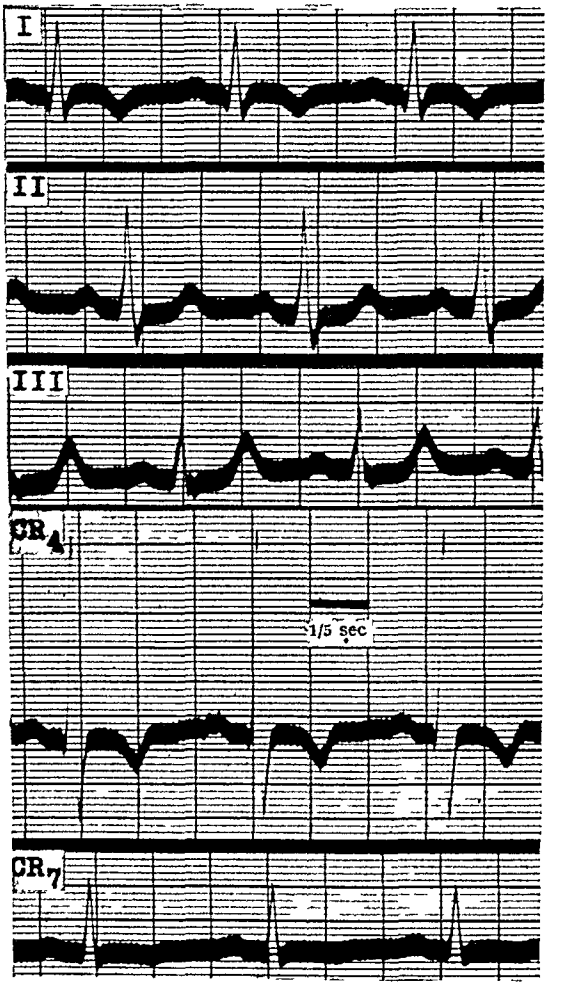


FIG. 128

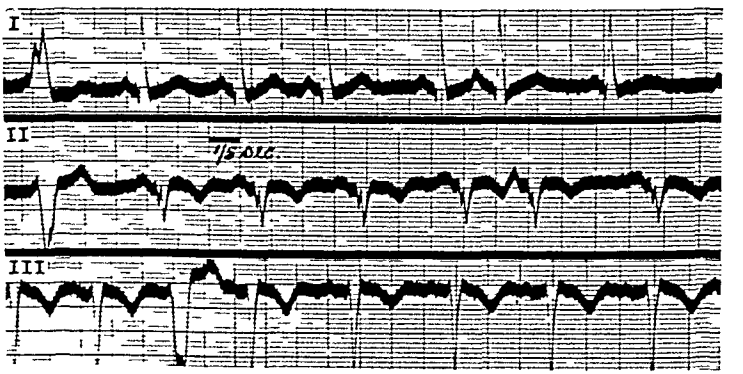


FIG. 129

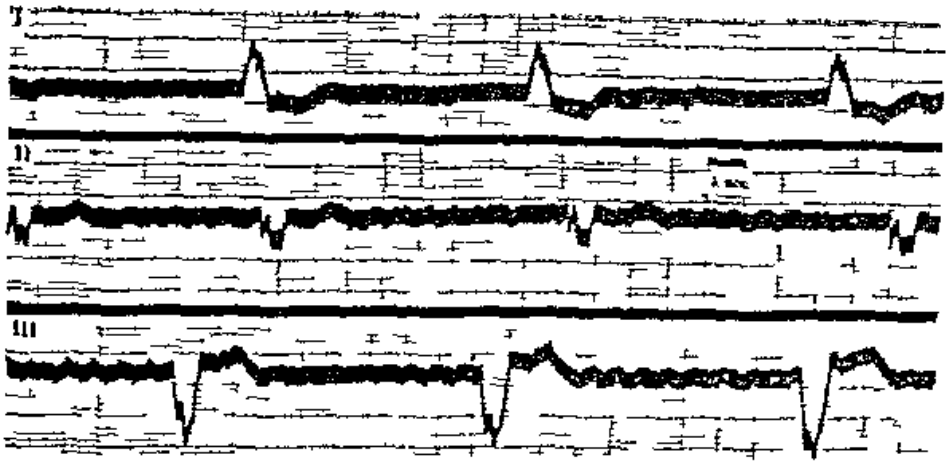


FIG 130

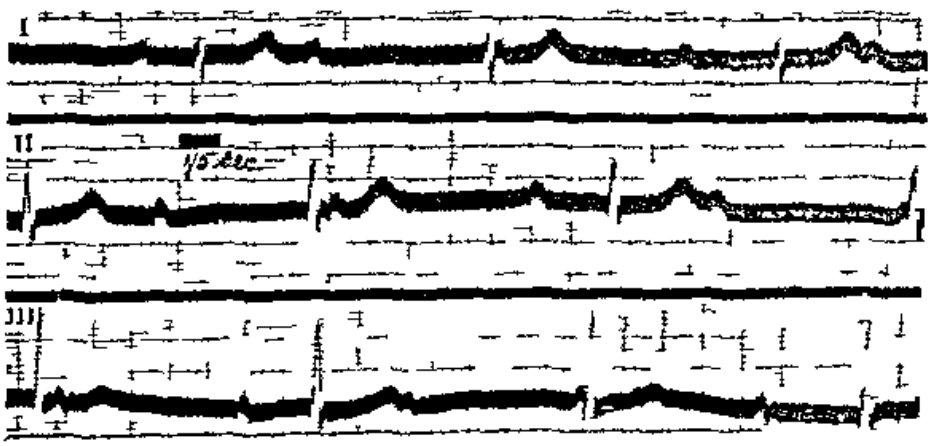


FIG 131

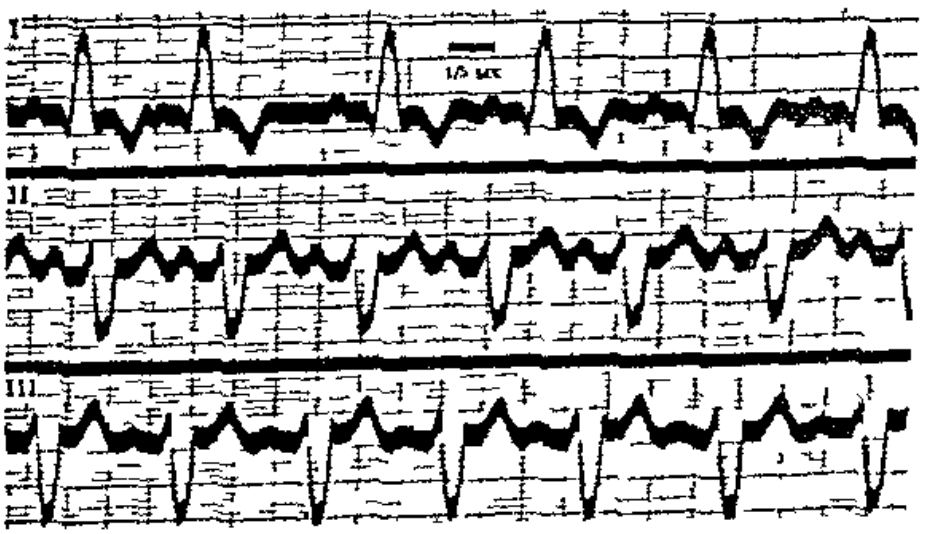


FIG 132

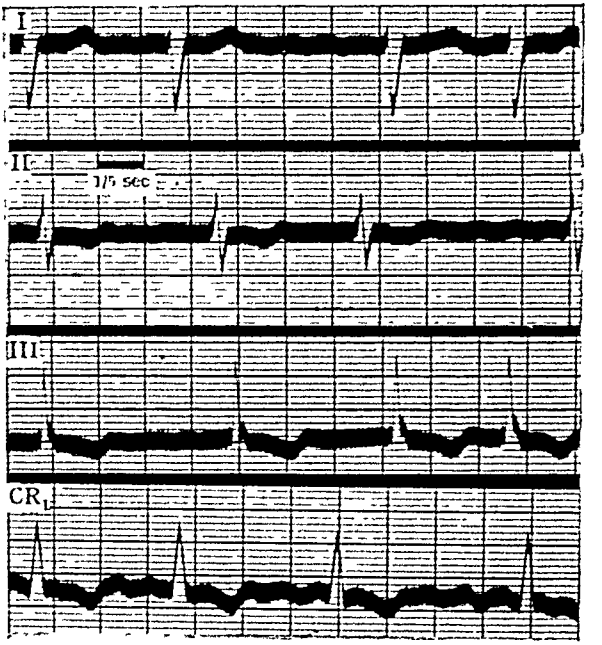


FIG. 133

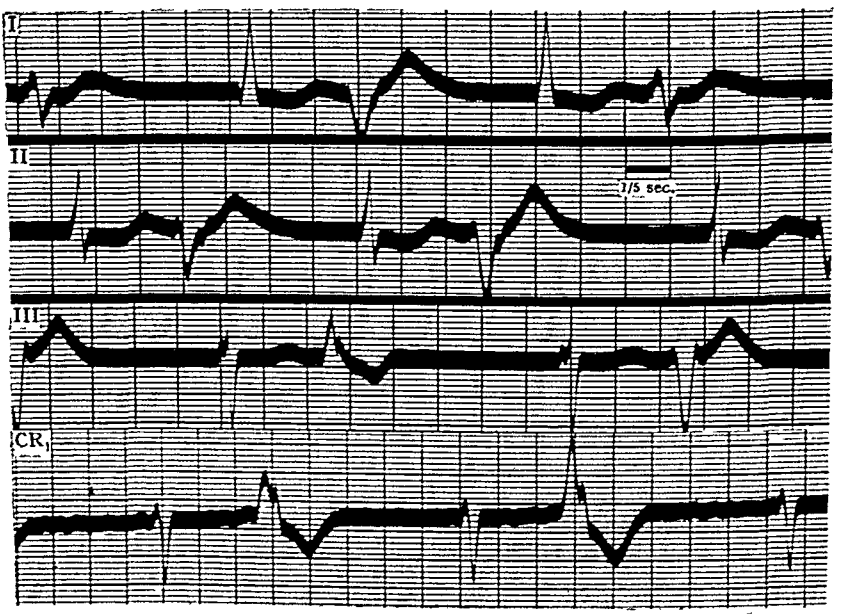


FIG. 134

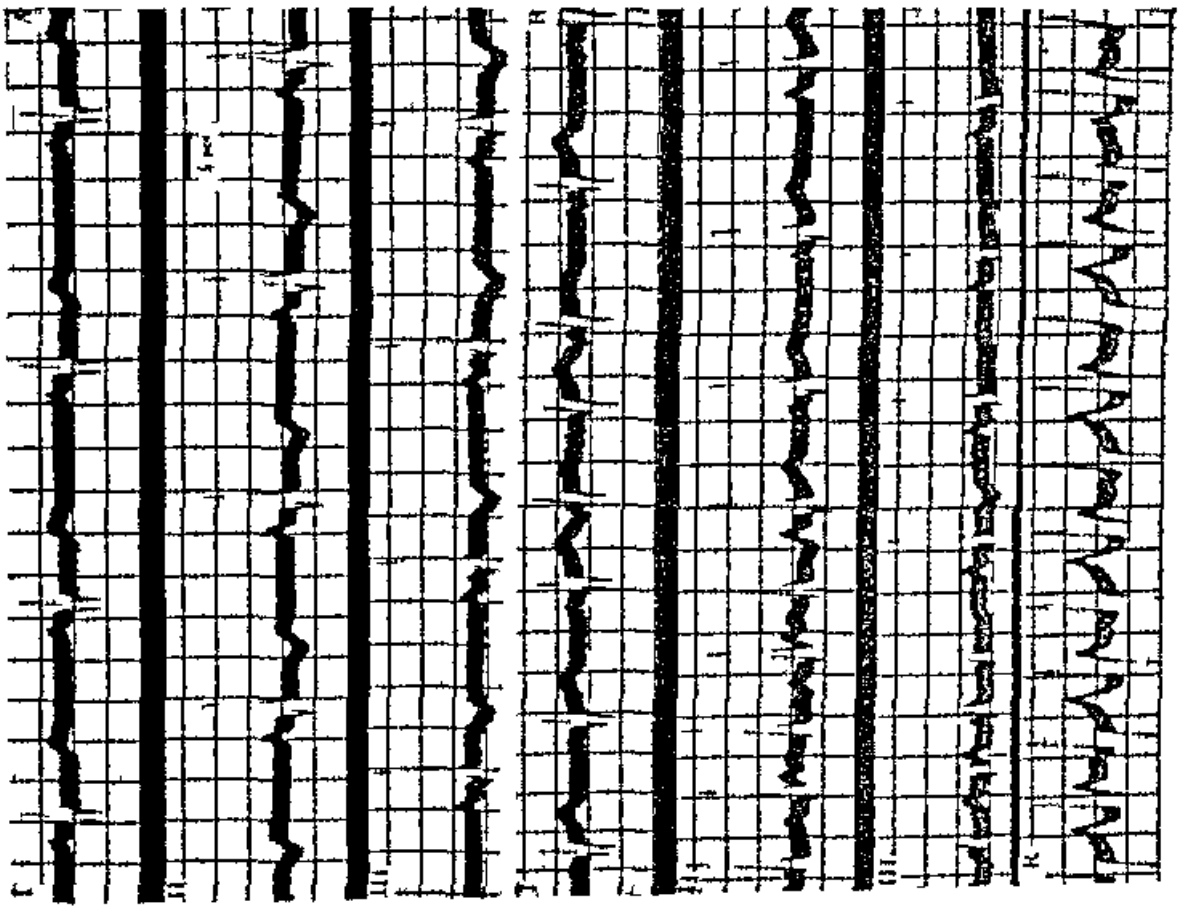


FIG 136

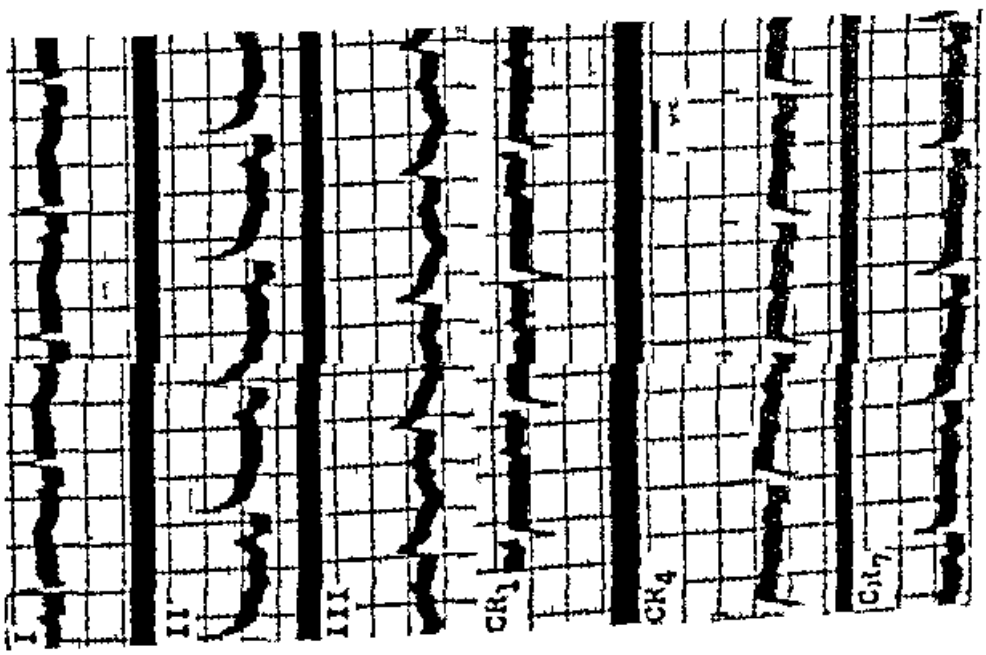


FIG 135

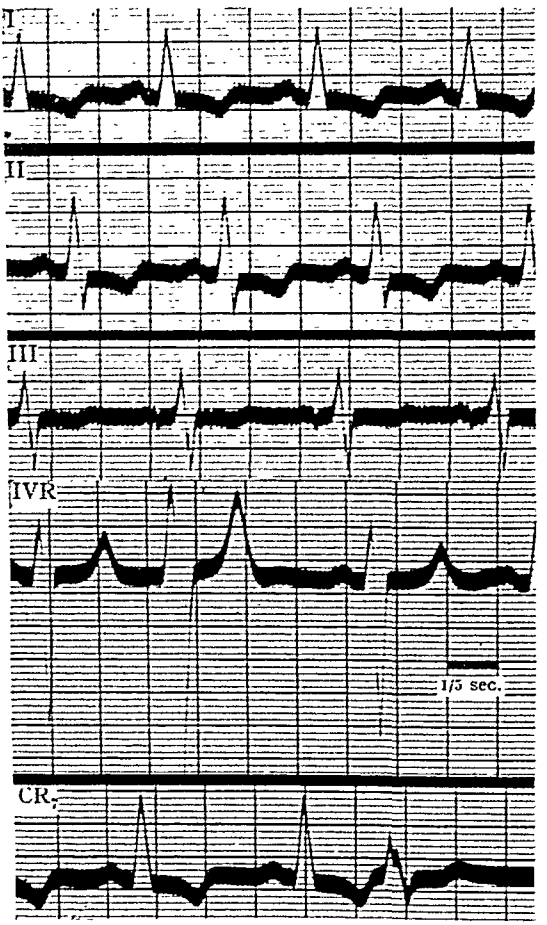


FIG. 137

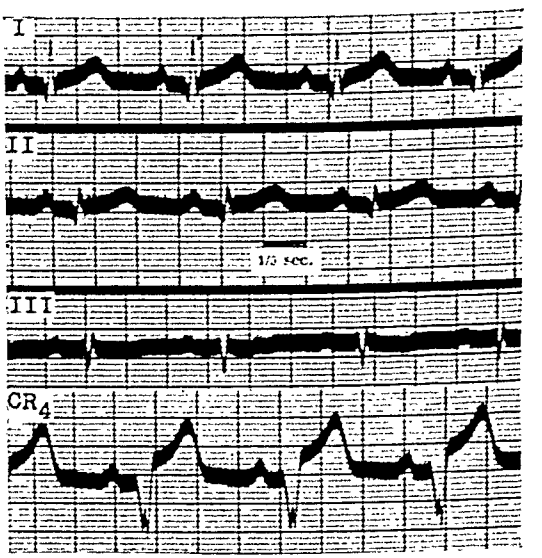


FIG. 138

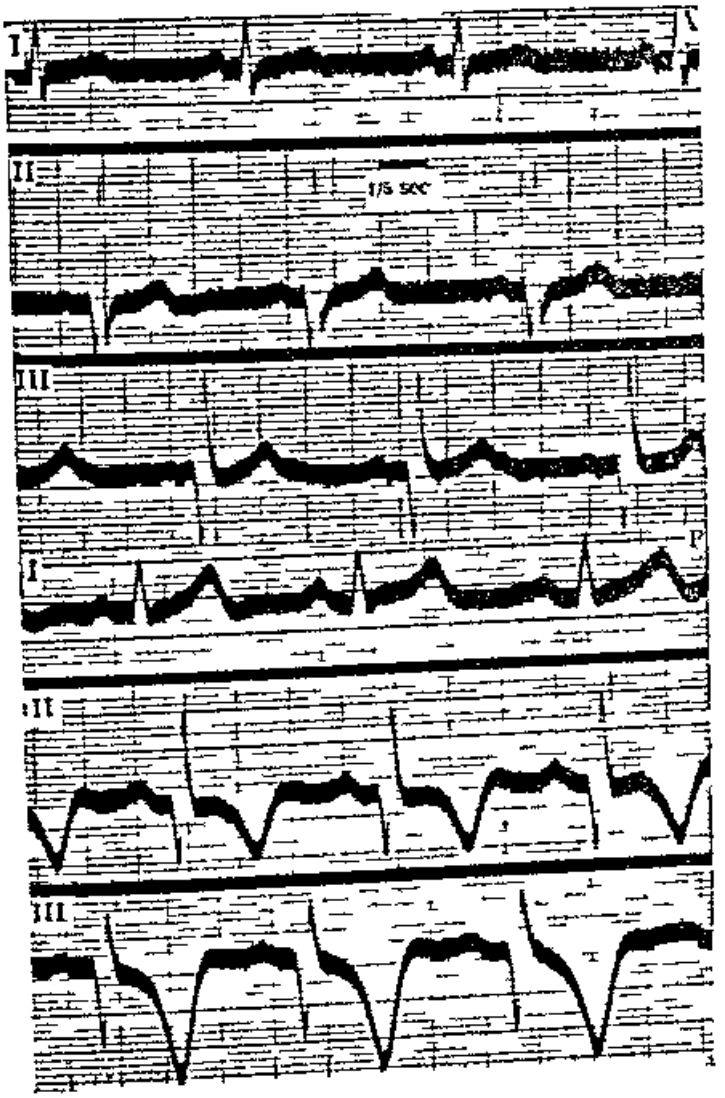


FIG 139

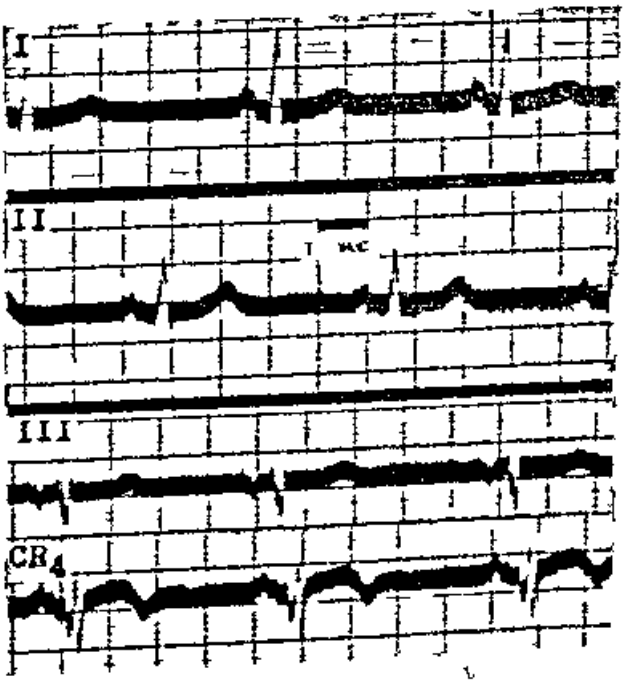


FIG 140

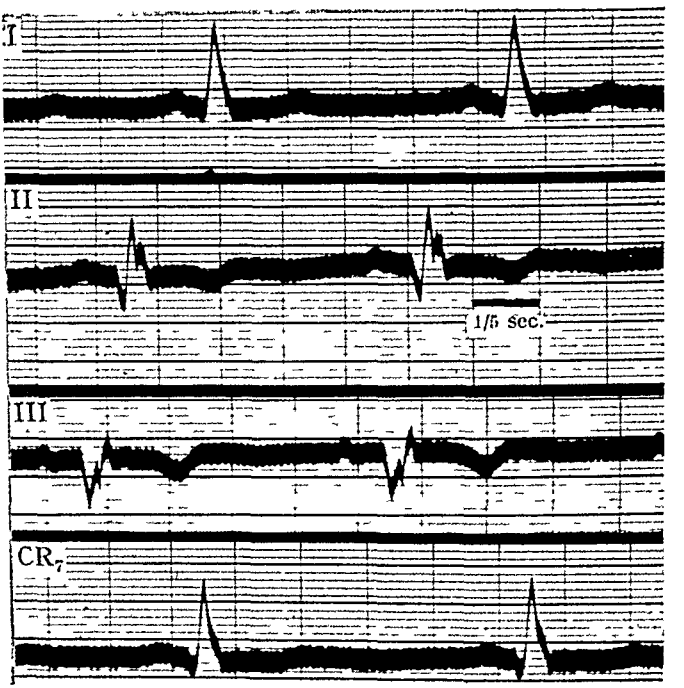


FIG. 141

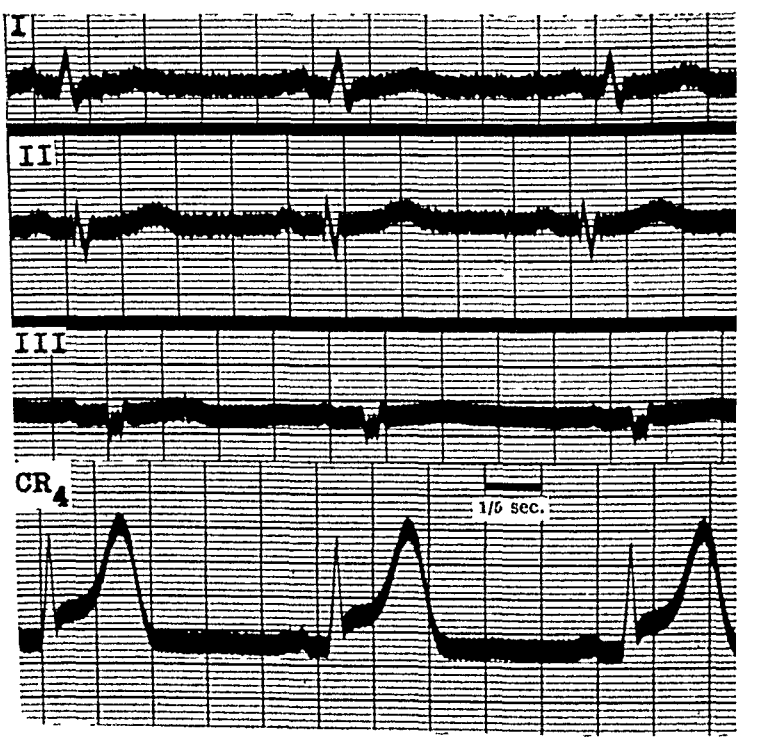


FIG. 142

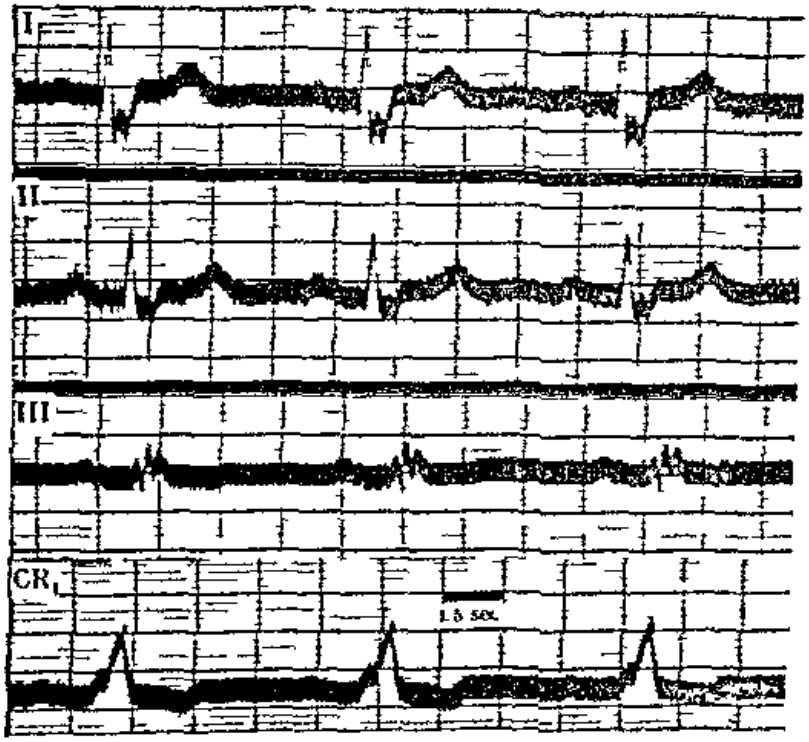


FIG 143

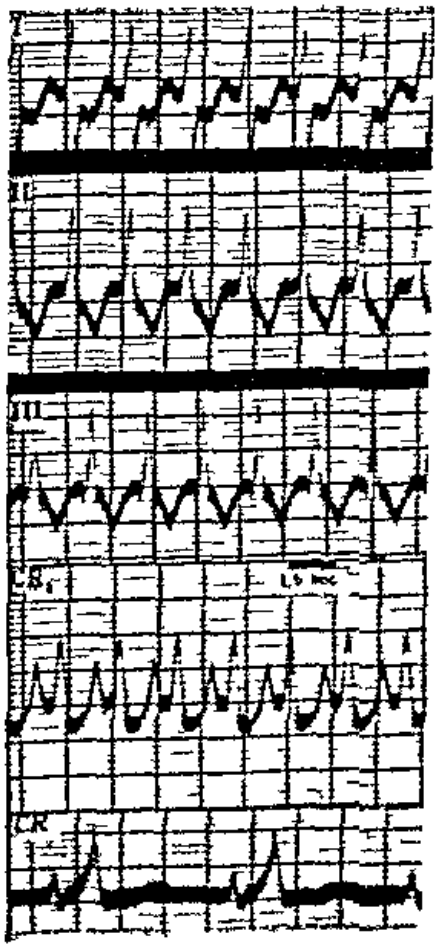


FIG 144

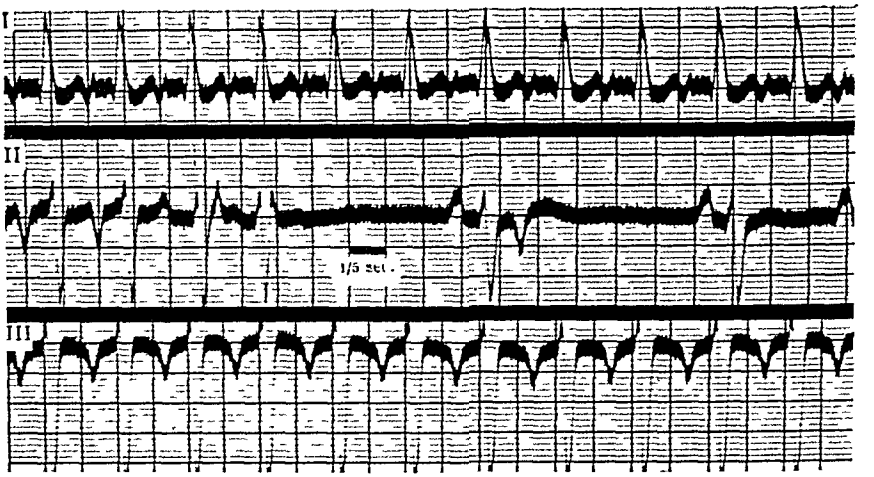


FIG. 145

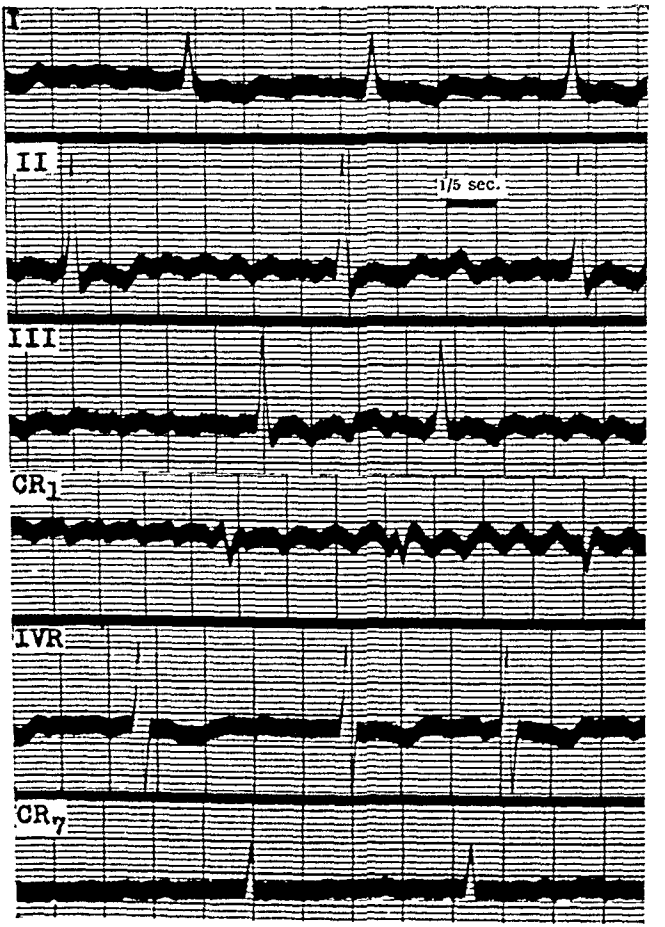


FIG. 146

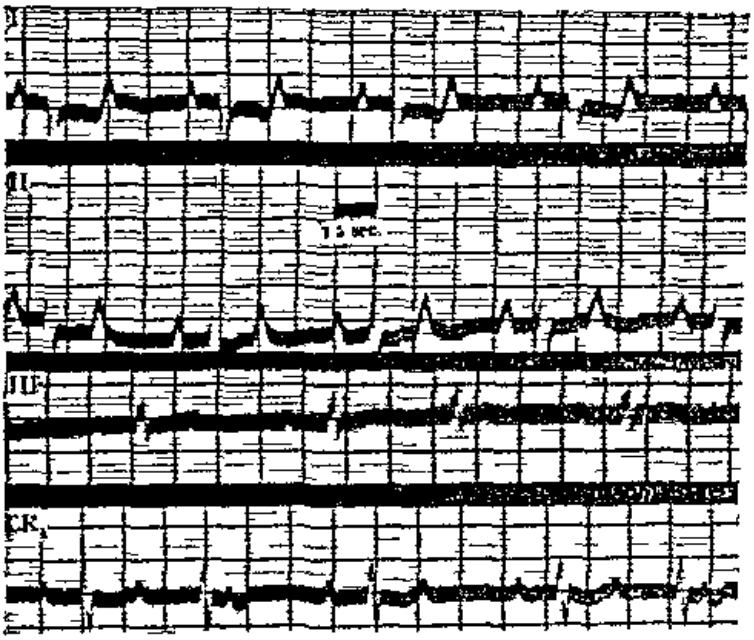


Fig 147

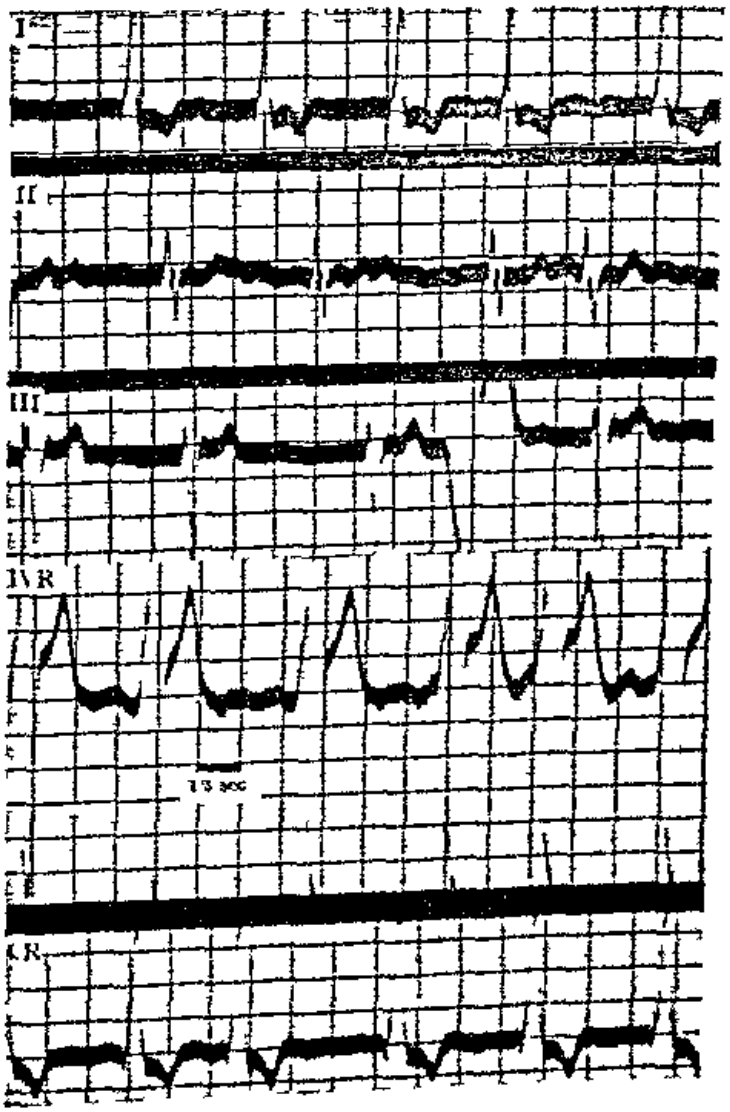


Fig 148

KEY TO TEST ELECTROCARDIOGRAMS

FIG. 111.—The ventricular rate is slow. The rhythm is irregular from 2 to 1 heart block. The P wave is small in III. The T wave is low in I and inverted in II and III. The Q wave is prominent in II and especially in III. The R–T segment is depressed in I and deviated upwards (Pardee sign) in II and III. *Recent cardiac infarction (posterior) with heart block.* (In this and in subsequent key legends the cardiographic diagnosis is given in italics).

FIG. 112.—The rate is normal. The rhythm is regular. The P–R period is normal. Slight R.A.D. The T wave is upright in all leads. The R–T segment is elevated in all leads, especially in I and CR_1. *Recent cardiac infarction (anterior).*

FIG. 113.—The rate is very slow. The rhythm is irregular from heart block and at times there is auricular standstill. No axis deviation. The T wave is inverted in lead I. *Heart block with sometimes auricular standstill from cardiac infarction (anterior).*

FIG. 114.—The rate is normal. The rhythm is regular. The P–R period is normal. No electrical axis deviation. The T wave is upright in all leads. *Physiological tracing.*

FIG. 115.—The rate is slow. The rhythm is irregular from complete heart block. The P waves are hardly visible in the limb leads and are best seen in CR_1. No axis deviation. The T wave is inverted in III and is low in CR_1. *Complete heart block.*

FIG. 116.—The rate is slow. The rhythm is irregular from complete heart block. L.A.D. Left bundle branch block. *Complete and bundle branch block.*

FIG. 117.—The rate is rapid. The rhythm is regular. The P–R period is full. R.A.D. The P wave is tall and spiky in II and CR_1. The T wave is inverted in III and CR_1. Splintering of the QRS with hardly an S wave in CR_1. *Right axis deviation and right ventricular preponderance in Fallot's congenital heart disease.*

FIG. 118.—The rate is very slow. The rhythm is irregular from auricular fibrillation where the " f " waves are hardly visible in the limb leads but are prominent in CR_1. QRS complexes wide. The T waves are inverted in the three limb leads. *Slow auricular fibrillation and bundle branch block.*

FIG. 119.—The rate is normal. The rhythm is abnormal from nodal extrasystoles. The P–R period is normal. L.A.D. The T wave is low in I and inverted in II and III. *Extrasystoles in cardiac infarction (posterior).*

FIG. 120.—The rate is slow. The rhythm is irregular in lead II from ventricular escape. No axis deviation. The T waves are upright in all leads. *Ventricular escape in sinus bradycardia.*

FIG. 121.—The rate is normal. The rhythm is regular. The P–R period is not prolonged. R.A.D. The P wave is bifid and the QRS wide. The T wave in lead III is inverted. *Mitral stenosis and auricular septal defect (Lutembacher's syndrome).*

FIG 122.—The rate is not rapid. The rhythm is irregular from auricular fibrillation. L A D. Prominent "f" waves. T wave inverted in lead I. *Auricular fibrillation in aortic incompetence and mitral stenosis.*

FIG 123.—The rate is a little slow. The rhythm is regular. The P–R period is *normal. The S wave is deep and wide in leads I and II.* The T wave is inverted in lead III. *Right bundle branch block.*

FIG 124.—The rate is normal. The rhythm is regular. The P–R period is slightly prolonged. R A D. The QRS is directed upwards in CR_1 and downwards in CR_7. The T wave is inverted in leads III and CR_1. *Right ventricular preponderance in pulmonary stenosis* (P M control).

FIG 125.—The rate is a little rapid. The rhythm is regular. The P–R period is *normal. No axis deviation. The P wave is inverted in leads II and III.* The T wave is diphasic in lead III. A U wave is seen in lead I. *A displaced pacemaker or parasitus rhythm.*

FIG 126.—The rate is normal. The rhythm is regular. The P–R period is short. No axis deviation. The T wave is inverted in leads II, III and CR_4, and is low in CR_7. *Temporary effects following an attack of paroxysmal tachycardia.*

FIG 127.—The rate is normal. The rhythm is irregular from auricular fibrillation *and alternating extrasystoles. Small voltage in lead I.* The f ' waves are more prominent in CR_1. The R–T segment is depressed in leads II and III. *Coupling effect in auricular fibrillation from digitalisation.*

FIG 128.—The rate is normal. The rhythm is regular. The P R period is normal. No axis deviation. The T wave is inverted in leads I and CR_4 and less inverted in CR. *Cardiac infarction (anterior).*

FIG 129.—The rate is a little rapid. The rhythm is irregular from auricular and nodal extrasystoles. The P–R period is normal. L A D. The T wave is inverted in leads II and III. *Cardiac infarction (posterior) and extrasystoles.*

FIG 130.—*The rate is very slow. The rhythm is irregular from* auricular fibrillation. L A D. The QRS complexes are wide and the T waves are written in the opposite direction to the QRS. *Auricular fibrillation and left bundle branch block.*

FIG 131.—The ventricular rate is slow. The rhythm is *irregular from complete* heart block. Slight R A D. The T wave is upright in all leads. *Complete heart block.*

FIG 132.—The rate is a little rapid. The rhythm is regular except for one auricular extrasystole in lead I. The P–R period is a little prolonged. L A D. The QRS complexes are wide and the T waves are written in the opposite direction to the QRS. *Left bundle branch block.*

FIG 133.—The rate is not rapid. The rhythm is irregular from auricular fibrillation. R A D. The T wave is diphasic in lead II and inverted in leads III and CR_1. The S wave is absent in CR_1. *Auricular fibrillation in mitral stenosis with pulmonary incompetence.*

FIG 134.—The rate is normal. The rhythm is irregular from auricular fibrillation and alternating extrasystoles. L A D. The T wave is low in most leads with

FIG 145.—The rate is very rapid in leads I and III but is slow in second half of lead II A–V dissociation is not apparent In the first isolated beat in lead II, an inverted P wave is placed in front of the T wave but does not initiate its own ventricular complex *Paroxysmal tachycardia (parasimus) with a blocked auricular extrasystole after slow sinus rhythm has been reinstated*

FIG 146.—The rate is normal The rhythm is irregular from auricular fibrillation No axis deviation The T wave is slightly inverted in leads I and CR_7 and more inverted in III II and IVR *Auricular fibrillation in constrictive pericarditis*

FIG 147.—The rate is normal At first sight the rhythm appears to be abnormal from heart block where the P seems to coincide with the T wave in CR_1 however the presence of two nodal extrasystoles suggests a sinus origin for the standard rhythm The P R period is slightly prolonged No axis deviation The R T segment is depressed in I *Slight but significant changes in a child with rheumatic fever*

FIG 148.—The rate is a little rapid The rhythm is irregular from auricular fibrillation and extrasystoles L A D The T wave is inverted in leads I and CR The R–T segment is raised in IVR *Auricular fibrillation in aortic incompetence*

PART II

PHONOCARDIOGRAPHY

INTRODUCTORY

HEART sounds and murmurs may be recorded by different physical devices but all the records shown here have been registered by the *string galvanometer* The chest piece consisted of a tambour whose taut elastic membrane forming *the diaphragm carried a metal* disc gummed to its inner surface Thence the sound waves were conducted through a glass tube (46 centimetres long) into a carbon microphone and an amplifier supplied by a 4-volt battery The initiated electrical current was led into one unit of a double string galvanometer and the movement of the fibre photographed A sound record has no value unless it is examined alongside some other record of the separate phases of the cardiac cycle , the electrocardiogram provides such a control, and when this was recorded by the second string of the galvanometer, both the electrocardiogram and the phonocardiogram were traced simultaneously on the same plate

Phonocardiography need not become a routine test in every case of heart disease, but it has proved indispensable in establishing a clinical classification for added heart sounds creating a triple and even a quadruple rhythm and in solving many problems connected with innocent as well as organic murmurs Should an auscultatory sign continue to be uncertain on clinical examination phonocardiography will help to explain it, and for this reason it is necessary to be acquainted with the test

HEART SOUNDS

When the phonocardiogram is traced alongside the electrocardiogram (Fig 149) in the way described the relation of the separate moieties of the two curves is as follows The auricular sound commences to be recorded at or towards the end of the P wave of the electrocardiogram The ventricular sound starts within the R–S period and is superimposed on the last phase of the auricular sound the duration of the auricular sound may be appreciated from a record taken in complete heart block (Figs 150 and 198) in which the whole of the auricular sound is recorded without interruption occasioned by the ventricular sound The second heart sound commences at a point corresponding with the end of the T wave, although its exact incidence should be judged in relation to the jugular tracing

SPLITTING OF HEART SOUNDS

It is incorrect to speak of duplication, reduplication, or doubling of the heart sounds. The first heart sound is made up of two components, the one produced by auricular systole and the other by ventricular systole , under no circumstance

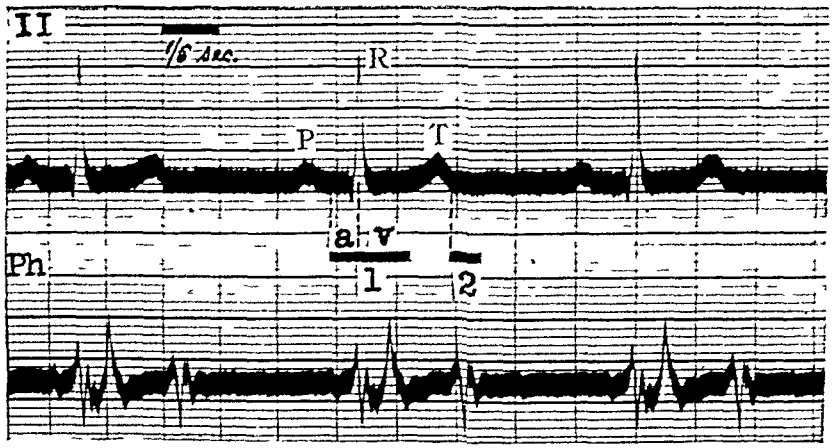


FIG. 149.—Normal phonocardiogram. The auricular sound (*a*) starts at the end of the P wave of the electrocardiogram, and the ventricular part of the first heart sound (*v*) coincides with the R wave. The second heart sound commences at the end of the T wave.

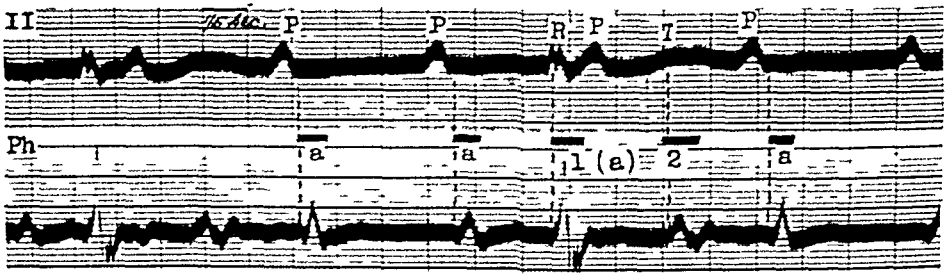


FIG. 150.—The isolated auricular sound (*a*) in complete A–V dissociation.

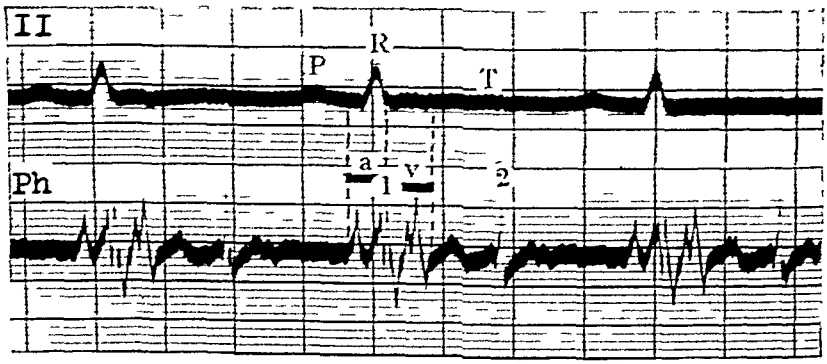


FIG. 151.—Splitting of the first heart sound. The auricular moiety (*a*) of the sound is relatively more prominent than the ventricular (*v*).

are these two components doubled but there is often splitting or separation of such components Similarly, the second heart sound produced by closure of the aortic and pulmonary valves is never doubled although it may again show splitting

Splitting of the first heart sound

If the first heart sound appears to display a dual nature on clinical auscultation it is the result of either slight separation of the auricular and ventricular components or the accentuation of one over the other Thus the mechanism is a form of splitting and no additional sound has been introduced The importance of this physical sign lies in the frequency with which it is mistaken for the presystolic murmur of mitral stenosis It is a common sign and is met with in three circumstances

FIG 152.—Splitting of the first heart sound The ventricular moiety (*v*) of the sound is more prominent than the auricular (*a*)

In health.—Splitting of the first heart sound (abbreviated Ĭ) in the mitral area is found commonly in healthy subjects at all ages No sign of heart disease can be identified with this auscultatory finding on clinical or radiological examination The electrocardiogram too is normal and the P R period is not prolonged The phonocardiogram is distinctive and shows an exaggeration either of the auricular sound wave the auricular type (Fig 151) or less commonly of the ventricular wave the ventricular variety (Fig. 152) The splitting shown by patients with hypertension also conforms with one of these two varieties identified with health

Splitting of the first heart sound may be told from a presystolic murmur by its abruptness (*r rup* compared with *thur rup*) by its site of greatest audibility over the xiphisternum and in that it is louder in the upright posture Splitting is a common finding in depression of the sternum probably on account of the proximity of the heart to the chest wall

With a lengthened P–R period.—It is natural to expect that moderate separation of the auricular and ventricular components of the first heart sound as an outcome of delay in the auriculoventricular conduction of the heart impulse will give

rise to splitting on auscultation (Fig. 153). A greater separation would produce two distinct sounds initiating triple rhythm. As the P–R period which causes splitting is only a little in excess of 0·2 second, any subject presenting this sign should not, on this count alone, be considered abnormal.

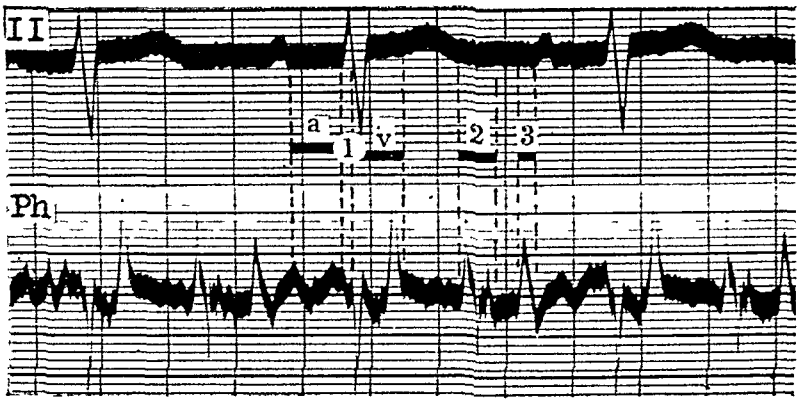


FIG. 153.—Splitting of the first heart sound in delayed A–V conduction which has prolonged the P–R period.

In bundle branch block.—Corresponding with a lengthening of the R–S period in the electrocardiogram of bundle branch block, is a delay in the appearance of the ventricular part of the first heart sound (Fig. 154). Since the P–R period is also frequently prolonged in bundle branch block, the common incidence of splitting of

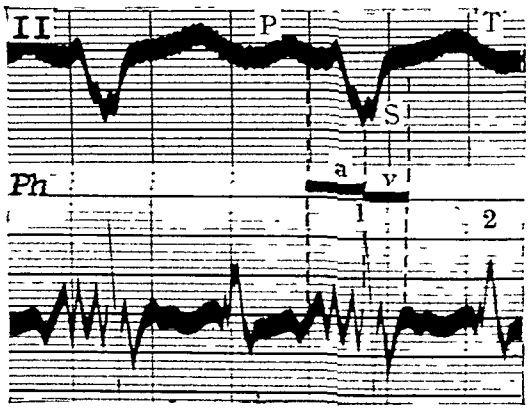


FIG. 154.—Splitting of the first heart sound in bundle branch block ; the ventricular part of the first heart sound (v) is delayed on account of the prolonged R–S period.

the first heart sound in this condition is readily understood. Indeed, this auscultatory sign in elderly subjects should direct attention to the possibility of a lesion of the bundle, and an electrocardiogram should be a routine test in such cases.

Splitting of the pulmonary second heart sound

When the second sound in the pulmonary area is accentuated it usually demonstrates splitting as well. It is improbable that the splitting is caused by the

pulmonary valve closing before the aortic for in the phonocardiogram (Fig 155) the second sound is no earlier than the normal but it lasts longer and this effect might be the outcome of vibration within the pulmonary artery after the closure of the valves Although the sign is commonly found in conditions associated with raised blood pressure in the pulmonary circulation notably mitral stenosis and congenital heart disease it is often present in healthy young subjects and therefore is without significance as a sign for by itself it cannot help in diagnosis

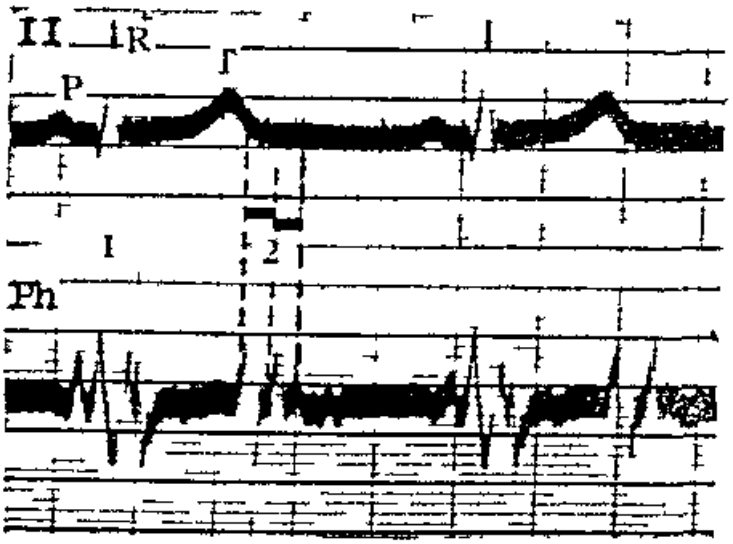


FIG 155 —Splitting of the second heart sound in the pulmonary area The second sound is prolonged and it has not started before its customary place The third heart sound is beyond the second heart sound

TRIPLE HEART RHYTHM

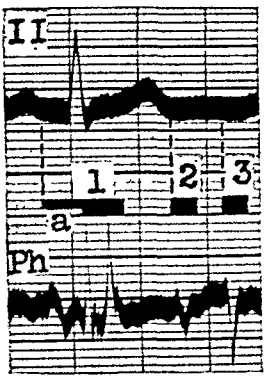
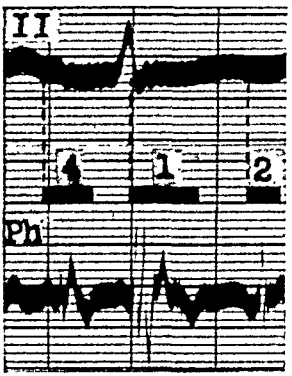
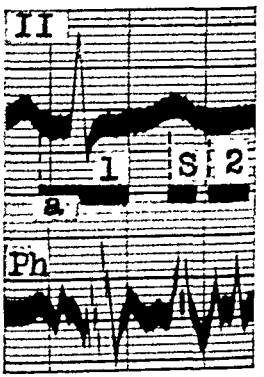
By triple rhythm is meant the cadence produced when three separate sounds recur in succeeding cardiac cycles It is a common auscultatory sign and in patients referred for examination of the heart at least it is as common as the familiar dual rhythm produced by the first and second heart sounds The appreciation of this common incidence of triple rhythm will come to us as soon as we adopt during clinical auscultation the simple form of self-catechism ' Do I hear more than two heart sounds ? "

A clinical classification of triple rhythm should deal with its cause in terms of heart disease and should deliberately exclude conjecture concerning the actual mechanism of the added sound When the position of the heart sound in the cardiac cycle is considered alongside the clinical state in patients with triple rhythm it is possible to place them in three groups (Fig 156 and Table III) As a rule except under the handicap of tachycardia the position of the extra sound can be told by auscultation aided by the clinical data and without phonocardiography although the test is necessary for the interpretation of a difficult case

Addition of the third heart sound

In this group the added sound appears early in diastole and is the third heart sound The auscultatory sign is found in young healthy subjects or in cases of

TABLE III. Comparing the characteristics of the three different types of triple heart rhythm.

Kinds of triple rhythm	*Addition of the third heart sound*		*Addition of the fourth heart sound*		*Addition of a sound in late systole*
Place of the added sound in cardiac cycle					
Cause	In health	*In right ventricular failure* Mitral stenosis Hypertension Congenital heart disease Thyroid toxaemia Emphysema ; primary pulmonary hypertension ; pulmonary embolism Anaemia Constrictive pericarditis Cardiac infarction Familial cardiomegaly	In delayed A–V conduction	*In left ventricular failure* Hypertension Aortic incompetence Cardiac infarction	In health
Site of maximal intensity	Internal to mitral area	Over displaced apex beat ; at xiphisternum	Internal to mitral area	At xiphisternum ; over displaced apex beat	Internal to mitral area
Effect of erect posture	Usually disappears	Persists	Persists	Persists	Persists
Effect of auricular fibrillation	None	None	Disappears	Disappears	None
Radiological features	Pulmonary artery full	Right heart enlarged	None	Left heart enlarged : pulmonary congestion	None
Probable mechanism	Vibration of ventricular wall from inrush of blood in early diastole		Auricular contraction	Vibration of hypotonic ventricle from auricular systole	Not known
Special features	In young subjects ; never after 40	Sound is followed by murmur in mitral stenosis	Usual cause of triple rhythm in bundle branch block	Double systolic impulse felt	Comparable with the innocent murmur in late systole

right heart failure Recognition of each kind comes from a regard of the site of maximal intensity of the sound, the effect of posture upon it and the presence or absence of heart disease

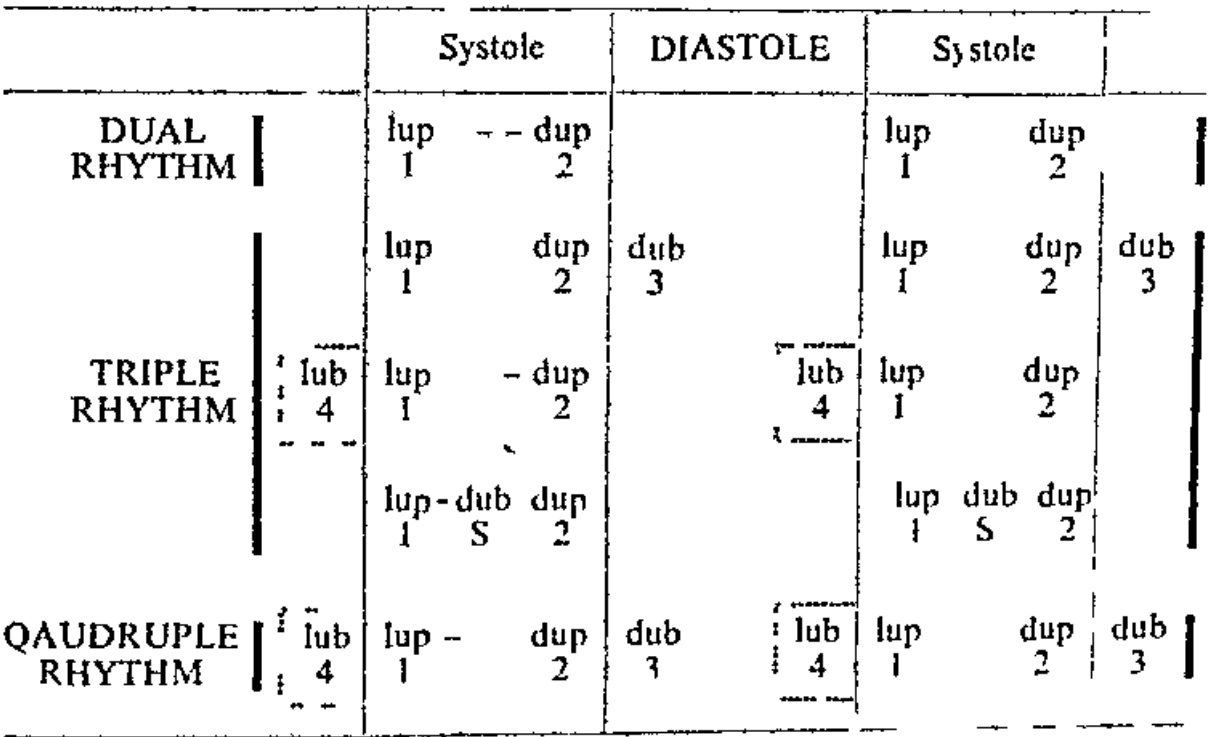


FIG 156.—Scheme showing the position of added sounds in triple and quadruple rhythm S is the added sound in late systole

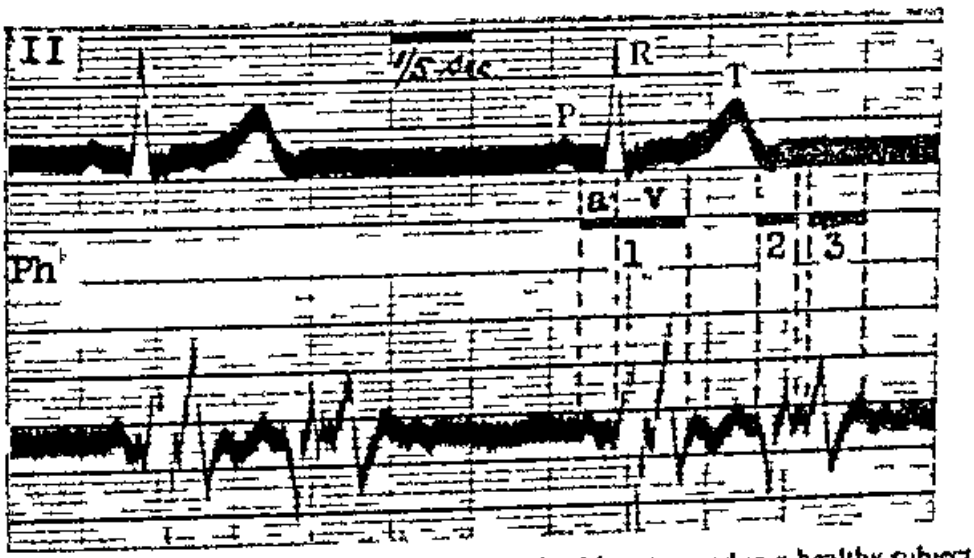


FIG 157.—Triple rhythm from addition of the third heart sound in a healthy subject

The third heart sound in health.—This innocent kind of triple rhythm (Fig 157) is best heard a little internal to the apex beat in the reclining posture, and it usually

becomes inaudible in the upright posture, especially on deep inspiration (Fig. 158) unless there is tachycardia causing it to persist ; it is a sign common in children and in youths, much less common after 25 years, and is seldom if ever heard after 40. If the third heart sound, elicited by auscultation in a subject over 40, is ever regarded as incidental, the deduction should not be made lightly, and only when the electrocardiogram shows no elongation of the P–R period (with or without bundle branch block), when cardioscopy shows no cardiac enlargement (minimal in heart failure from cardiac infarction, emphysema, constrictive pericarditis, and thyroid toxaemia), and when the phonocardiogram has demonstrated an obvious third heart sound. When such rules are applied, it is likely that very few, if any, instances of triple heart rhythm from addition of the third heart sound will be found in healthy subjects over the age of 40. It is this truth which invests such importance in this auscultatory sign. On cardioscopy slight prominence of the

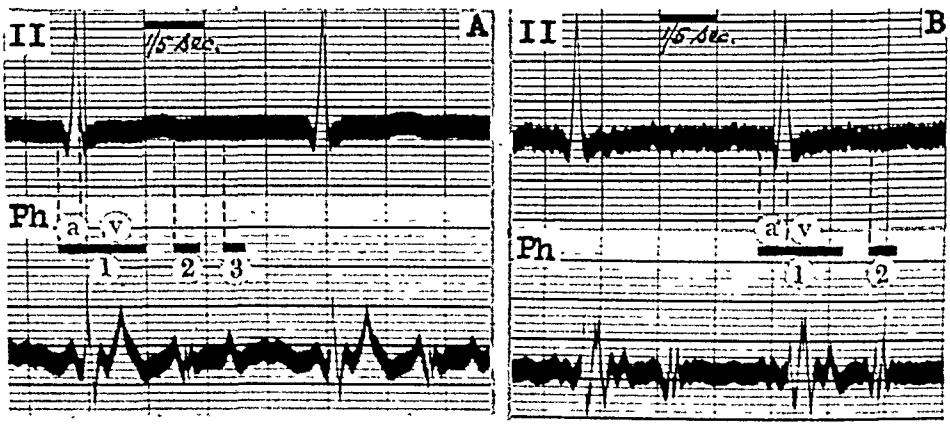
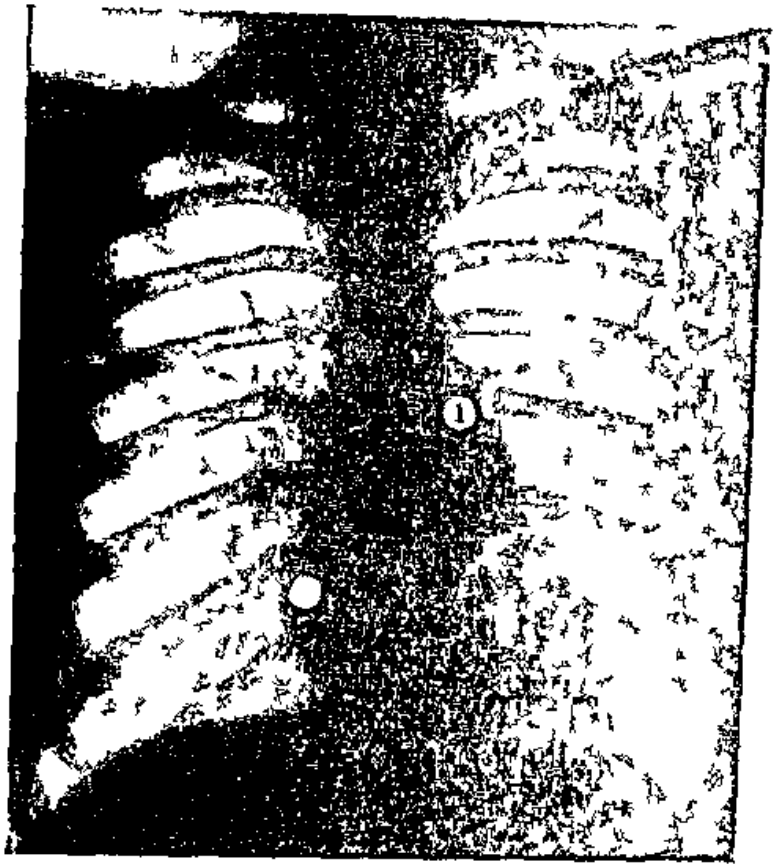


FIG. 158.—Triple rhythm from addition of the third heart sound in a healthy subject in the reclining posture (A), disappeared in the upright posture (B).

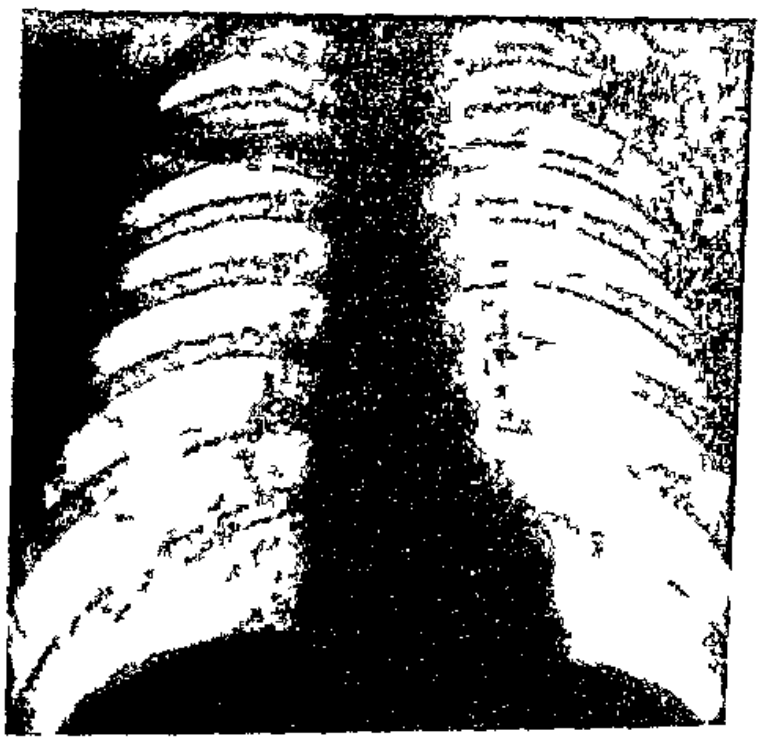
pulmonary artery is often present in subjects showing this triple rhythm (Fig. 159), and the T wave in the CR_1 cardiogram is frequently inverted ; such might be regarded as the triad which indicates *physiological right heart preponderance.*

The third heart sound in right ventricular failure.—Here the third heart sound is loudest over the displaced apex beat ; it persists with the patient in the upright posture, and there is evidence of disease which has caused enlargement or failure of the right heart, such as mitral stenosis, hypertension, thyroid toxaemia, congenital heart disease, emphysema, pulmonary embolism, primary pulmonary hypertension, constrictive pericarditis, anaemia, cardiac infarction, or familial cardiomegaly. This kind of triple rhythm is not limited to young subjects as is the innocent variety, and it bears repeating that a third heart sound in a patient over 40 indicates right heart failure, and one of the enumerated causes should be sought to account for it.

This triple rhythm is common in *mitral stenosis* (Fig. 160) and the addition of auricular fibrillation does not affect it (Fig. 161). In lone auricular fibrillation where the heart is not enlarged the third heart sound is not exhibited. Frequently



(A)



(B)

FIG. 159.—Fulness of the pulmonary artery (1) and the right auricle (2) in (A) is commonly associated with triple rhythm from addition of the third heart sound in health while in dual rhythm the pulmonary bay (1) is empty as in (B)

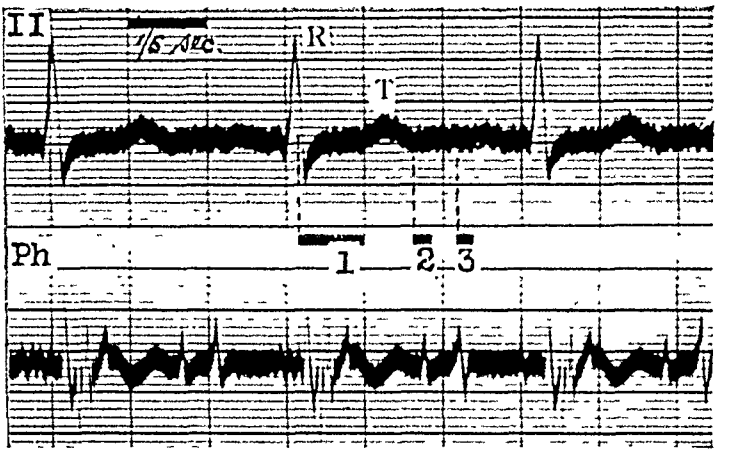


FIG. 160.—Mitral stenosis. Triple rhythm due to addition of the third heart sound ; this sound is followed by a mid-diastolic murmur.

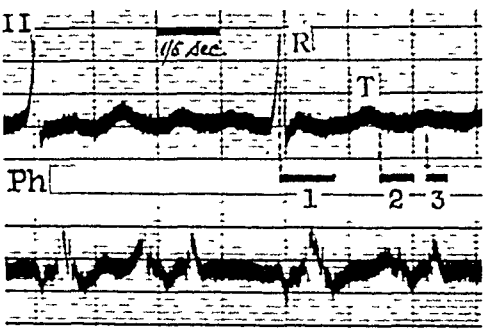


FIG. 161.—Auricular fibrillation in mitral stenosis. The third heart sound is prominent.

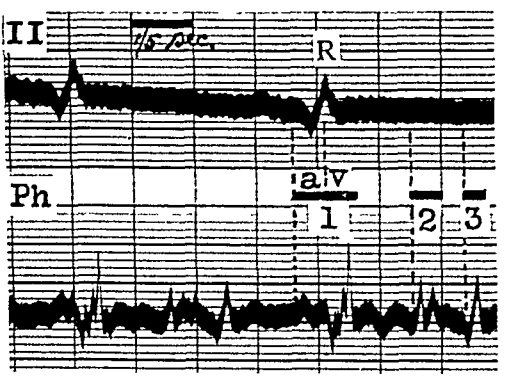


FIG. 162.—Hypertensive heart failure with prominent third heart sound.

the clear third heart sound in mitral stenosis gives way to the characteristic mid diastolic murmur which is contiguous with the third heart sound

When the left ventricle fails in *hypertension a triple rhythm due to* the addition of the fourth heart sound is commonplace but when the right heart has failed it is the third heart sound which often creates the triple rhythm Although the former variety may give way to the latter is the disease progresses the triple rhythm initiated by the addition of the third heart sound might be the first to appear in many patients with hypertensive heart failure (Fig 162) *The rhythm* may disappear on clinical auscultation for a time during treatment by rest and mercurial diuretics

Triple rhythm in thyroid toxaemia due to the addition of the third heart sound means that enlargement of the right heart and pulmonary artery has been a

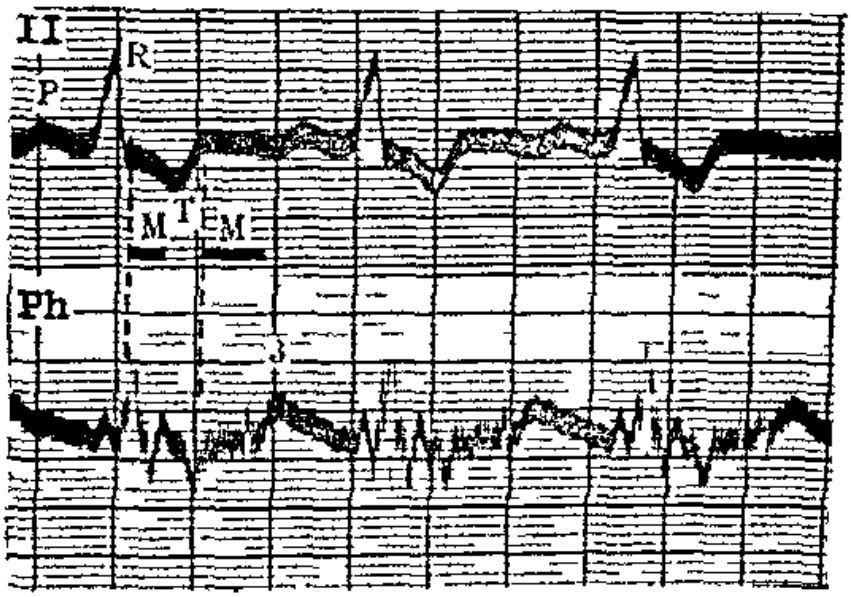


FIG 163.—Auricular septal defect Apart from systolic and diastolic murmurs (M) there is triple rhythm from addition of the third heart sound (3) which follows on the early diastolic murmur

complication of the condition Cardiac enlargement is not present in those cases with thyroid disease which show dual rhythm

Not all cases of *congenital heart disease* show this kind of triple rhythm but in those that do great enlargement of the right heart is a feature Auricular *septal defect* (Fig 163) *is a congenital anomaly* which almost invariably *initiates* triple rhythm

Clinical heart failure with conspicuous enlargement of the right heart is rare in *emphysema*, but failure can be presumed present if *triple rhythm* is found in a patient with emphysema (Fig 164) The finding of this auscultatory sign is an *important event in* such a case because it signifies that the patient cannot survive for many months The same triple rhythm may be heard in the uncommon condition of *primary pulmonary hypertension*

Triple rhythm from addition of *the third heart* sound is a valuable supporting sign in a patient taken suddenly ill with severe shortness of breath which suggests

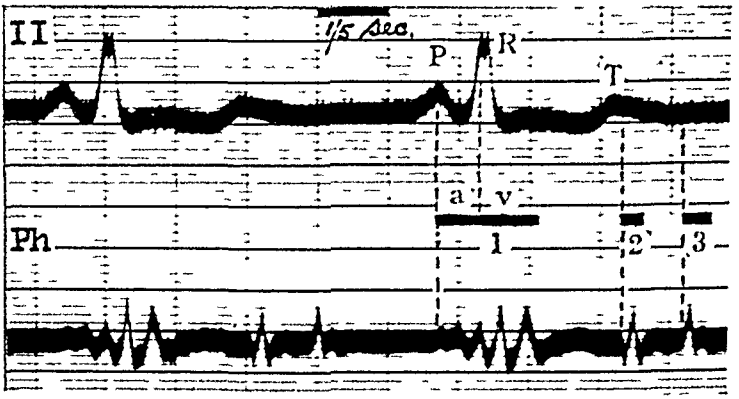


FIG. 164.—Emphysema and heart failure. The third heart sound is prominent.

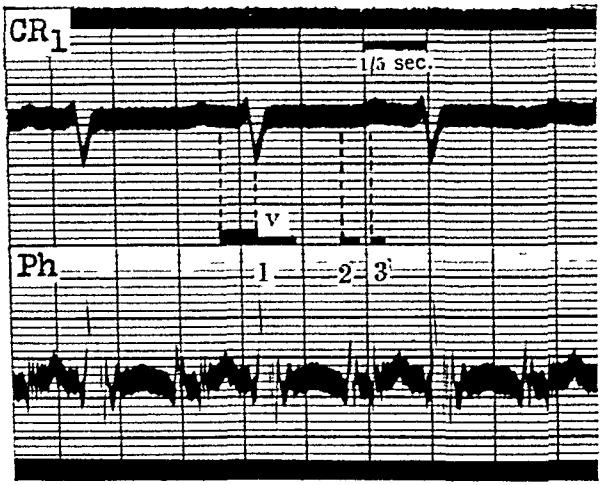


FIG. 165.—Constrictive pericarditis. Triple rhythm from addition of the third heart sound.

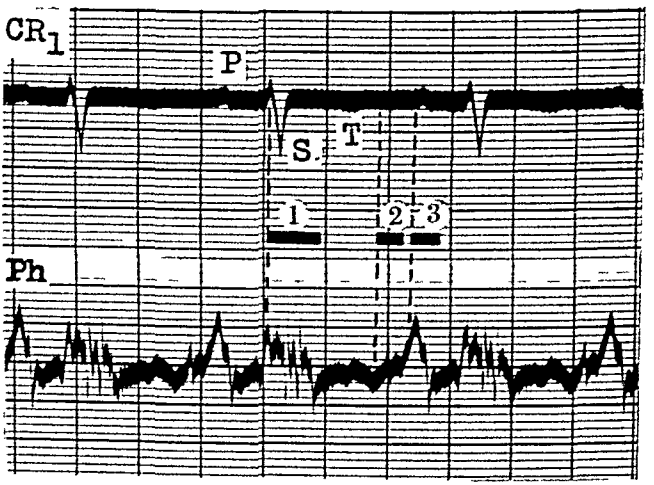


FIG. 166.—Anaemia in which there was much enlargement of the heart with triple rhythm from addition of the third heart sound.

the diagnosis of *pulmonary embolism* Indeed, it supplies the most reliable clue in the recognition of this critical illness apart from electrocardiography

The third heart sound is always present in *constrictive pericarditis* which causes symptoms (Fig 165), and for this reason it is a sign of great importance when such a diagnosis is entertained

When the heart is enlarged in *anaemia* a triple rhythm from addition of the third heart sound is a common finding (Fig 166)

Cardiac infarction may also cause this kind of triple rhythm and it is a noticeable sign in *familial cardiomegaly*

Addition of the fourth heart sound

Since the adventitious sound in this form of triple rhythm occupies a place later in diastole than that occupied by the third heart sound it may be called the fourth

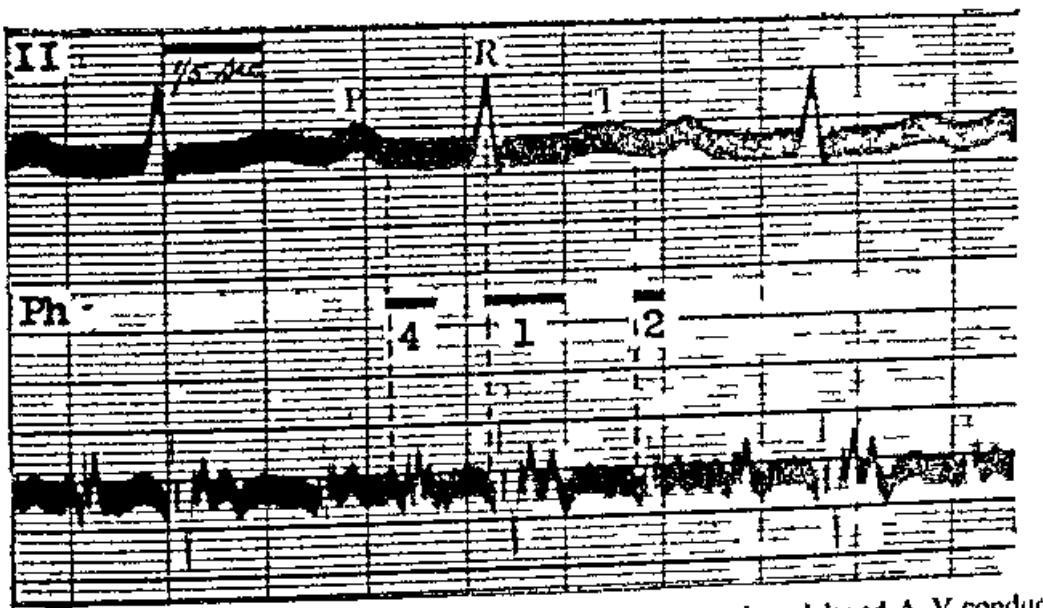


Fig 167 —Triple rhythm from addition of the fourth heart sound in delayed A V conduction The fourth heart sound (in this case the auricular sound) starts at the end of the P wave

sound and it immediately precedes the first heart sound It only precedes how ever, the *ventricular* moiety of the first heart sound because it occurs during auricular systole In this variety of triple rhythm also, there are two classes In the first, the supernumerary sound is produced by auricular systole and appears only when auriculoventricular conduction is delayed In the second, although the sound is produced in or by the left ventricle affected by failure regulated con traction of the auricle is necessary for its production Both varieties therefore are not heard in auricular fibrillation

The fourth heart sound in delayed A–V conduction —In healthy subjects the sound produced by auricular systole is easily recorded by the phonocardiograph, but its proximity to the sound of ventricular systole hinders the appreciation by clinical auscultation of these separate moieties of the first heart sound When A–V conduction is delayed and the P–R period of the electrocardiogram is prolonged to 0 22 second, or over, it is usually possible especially in young

subjects, to hear both auricular and ventricular sounds which along with the second heart sound, create a triple rhythm (Fig. 167). This triple rhythm, best heard a little internal to the mitral area and persisting in the upright posture, does not itself tell of heart disease because some 50 per cent of the subjects presenting it can be counted as healthy. Occasionally the auricular sound is so separated from the ventricular sound on account of the lengthened P–R period

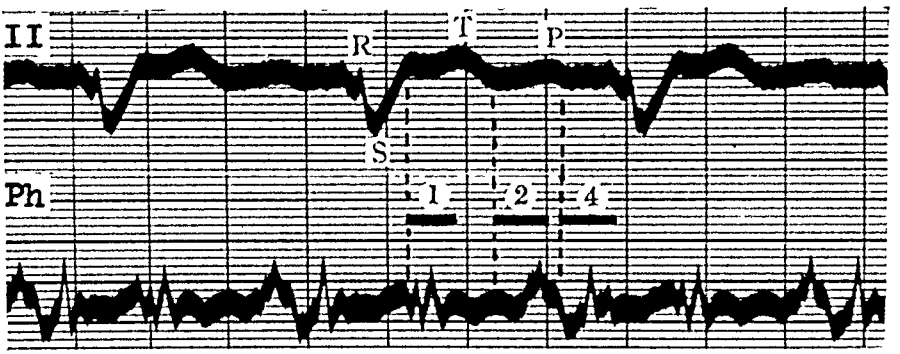


FIG. 168.—A lengthened P–R period in bundle branch block producing triple rhythm from addition of the fourth heart sound which appears so soon after the second heart sound as to make it appear clinically as triple rhythm from addition of the third heart sound.

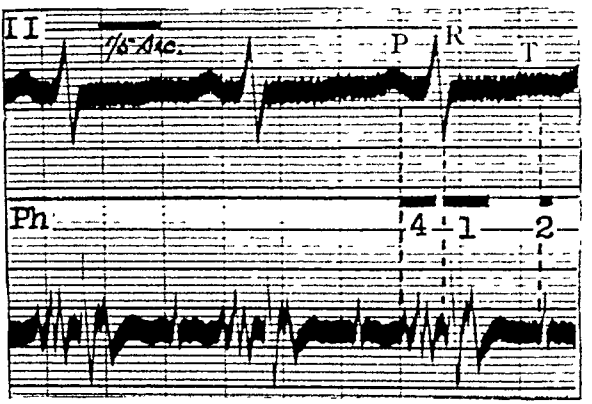


FIG. 169.—Hypertensive heart failure. Triple rhythm from addition of the fourth heart sound; this sound starts before the end of the P wave.

that it approximates the second heart sound (Fig. 168) and so may be mistaken for the third heart sound and its corresponding triple rhythm.

The fourth heart sound in left ventricular failure.—The extra sound from lengthening of the P–R period commences at the end of the P wave, but the sound in left ventricular failure commences earlier and often at a point corresponding with the summit of the P wave or even earlier. *Hypertension* is the usual cause of left ventricular failure initiating this form of triple rhythm (Fig. 169) although it is met with in some cases of *aortic incompetence* (Fig. 170). *Cardiac infarction* may sometimes initiate this kind of triple rhythm on its own, although it is commoner when it precipitates heart failure in hypertension. The abnormal rhythm

is sometimes best appreciated over the displaced apex beat, but usually it is best heard at the xiphisternum

During tachycardia the places occupied by the third and fourth heart sounds coincide producing a summation effect (Fig 171)

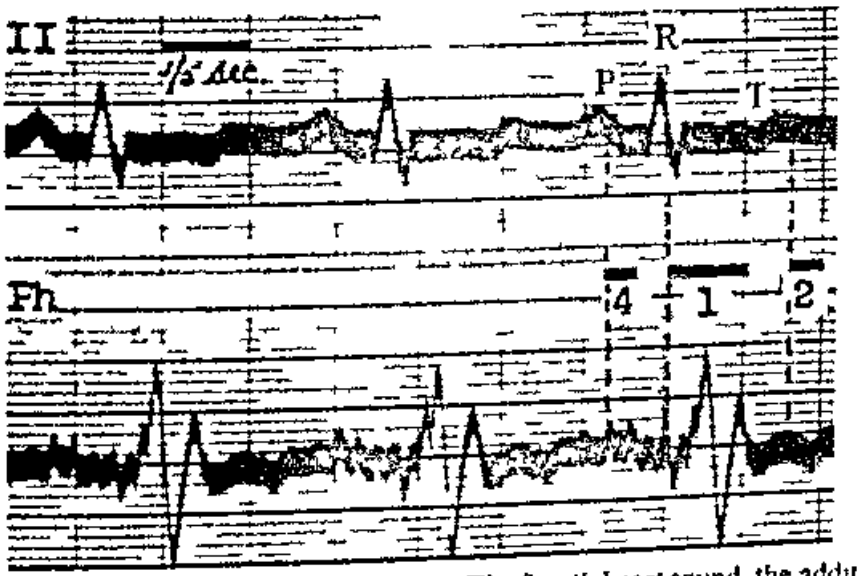


Fig 170 —Aortic incompetence and heart failure The fourth heart sound the addition of which produced a triple rhythm, commences before the end of the P wave

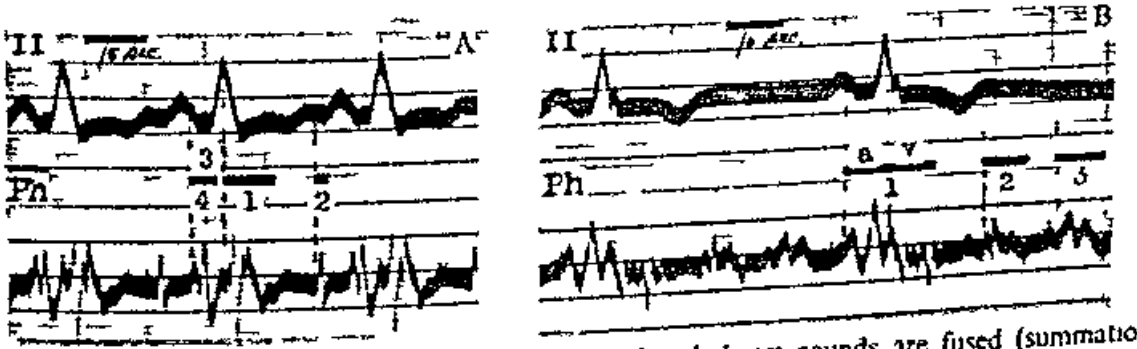


Fig 171 —During tachycardia (A) the third and fourth heart sounds are fused (summation effect) The sounds are separated in (B) when tachycardia has subsided From a patient with hypertensive heart failure

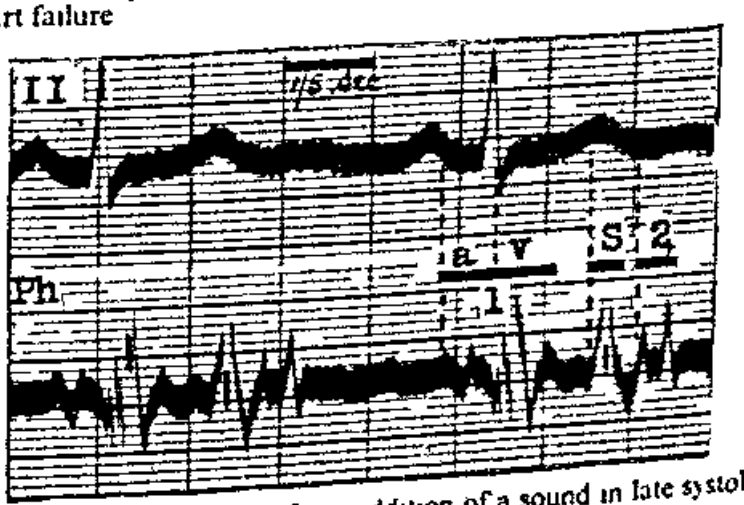


Fig 172 —Triple rhythm from addition of a sound in late systole

Addition of a sound in late systole

This kind of triple rhythm (Fig. 172) is likely to be mistaken for the one arising from addition of the third heart sound, but careful auscultation should readily localize the supernumerary sound in front of and not following the second sound. This auscultatory sign is uncommon, but when found it is not evidence of heart disease for the subjects presenting it are healthy. Its position in systole corresponds with that of the *late systolic murmur* which is also an innocent finding.

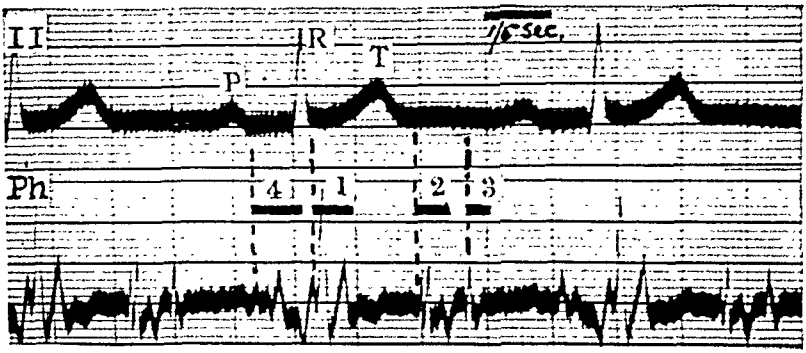


FIG. 173.—Quadruple heart rhythm. The third heart sound is present because of mitral stenosis, and the fourth heart sound is added because the P–R period is prolonged.

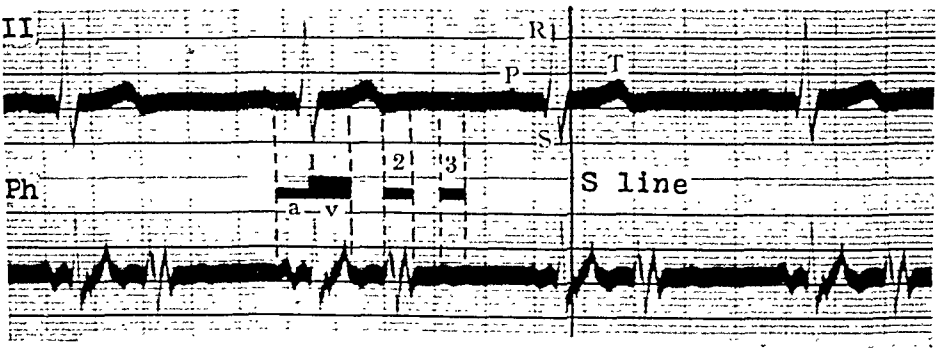


FIG. 174.—The S line is drawn through a point in the electrocardiogram which marks the finish of the S wave. It is an important landmark in the phonocardiogram.

QUADRUPLE HEART RHYTHM

There are a few ways in which the main or lesser varieties of triple rhythm may combine to produce a quadruple rhythm. The commonest is the combination of a delay in A–V conduction with one of the causes of right heart failure (Fig. 173).

HEART MURMURS

In the graphic interpretation of heart murmurs another landmark in the phonocardiogram has to be described in relation to the electrocardiogram. It is a line drawn from a point marking the end of the S wave in the electrocardiogram

to meet the sound record I have named this the S line (Fig 174) It is certain that a murmur starting at this line occurs during the early phase of ventricular systole, and one starting before it commences during auricular systole

To distinguish between the graphic representation of heart sounds and heart murmurs attention should be paid to the frequency of the vibrations These are infrequent and coarse in the case of heart sounds and frequent and finer in the case of murmurs (Fig 175)

THE INNOCENT MITRAL SYSTOLIC MURMURS

When the clinical cardioscopic and cardiographic findings in a series of healthy subjects with innocent murmurs in the mitral area were studied it was found that *they belonged to one of five groups*

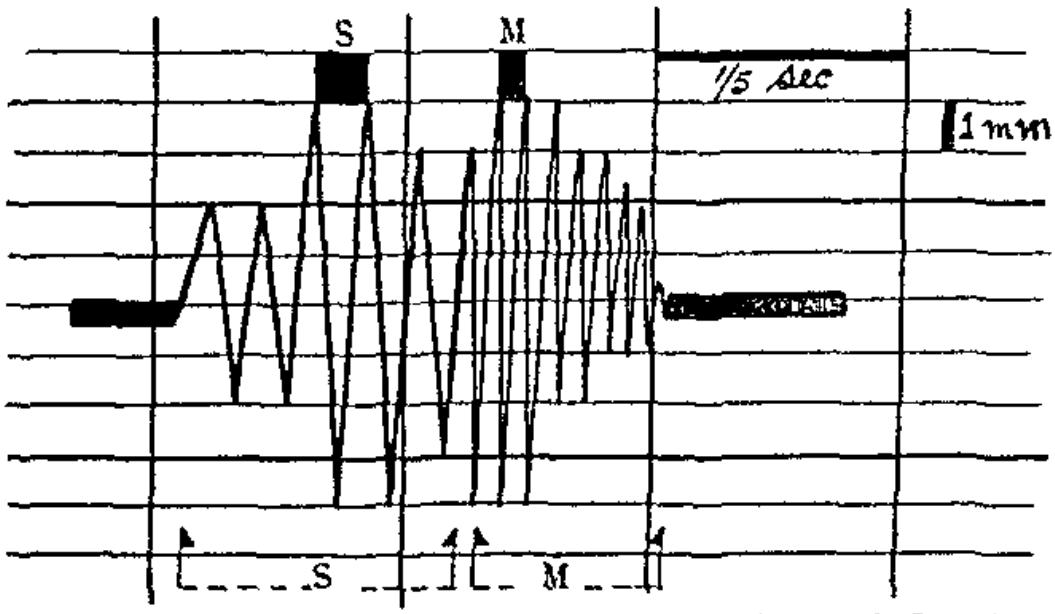


FIG 175 —Diagram showing the coarse vibrations of the heart sound (S) and the finer vibrations of the murmur (M)

The murmur of reclining posture

The murmur in subjects forming this group is not loud and on this account it sometimes disappears to auscultation on deep inspiration It is blowing in *character The murmur is louder in the reclining posture* when a similar murmur develops in the pulmonary area In some instances the pulmonary murmur is audible in the upright posture in this event it is louder than the mitral murmur when the reclining posture is assumed The murmur of reclining posture is common in young *subjects less common after* 30 *and* unknown after 40 In the phonocardiogram (Fig 176) the murmur is mid systolic in time and commences some distance beyond the S line

The murmur of upright posture

In this instance, too, the murmur is not loud and on this account it sometimes disappears to auscultation on deep inspiration It is blowing in character Although distinctly audible in the reclining posture the murmur is better heard *in the upright position* It is common in young subjects, less common after

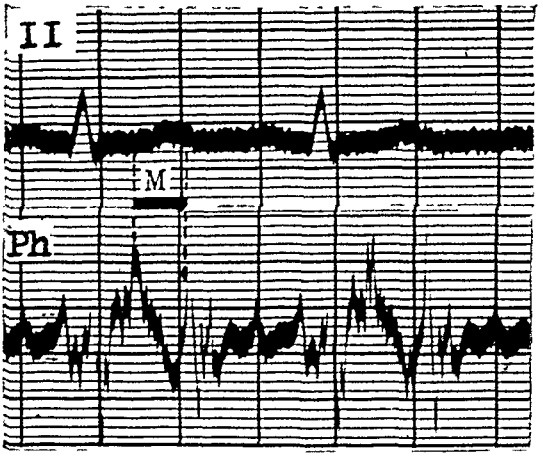


FIG. 176.—The innocent systolic murmur of reclining posture. The murmur (M) starts some distance after the S line and in mid-systole.

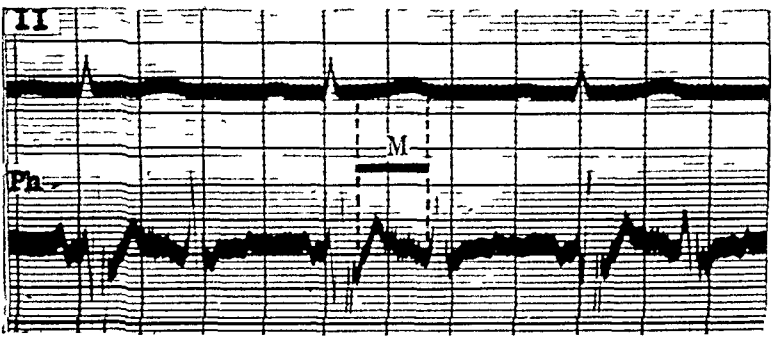


FIG. 177.—The innocent systolic murmur of upright posture. The murmur (M) starts in mid-systole and some way beyond the S line.

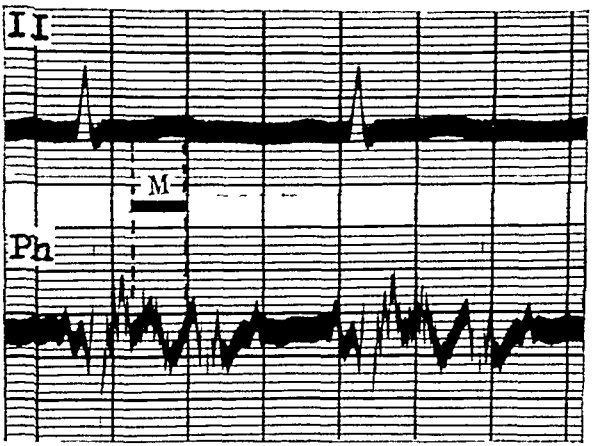


FIG. 178.—The loud variety of innocent systolic murmur. The murmur (M) starts in mid-systole and some way beyond the S line.

30, and is unknown after 40 In the phonocardiogram (Fig 177) the murmur is mid-systolic in time as in the previous group and commences some distance beyond the S line

The loud variety

This murmur is also confined to young subjects, and although loud its character is blowing or whiffy suggesting the high frequency of the vibrations which initiate it Posture affects its distribution more than its intensity so that in the upright position it can be heard far into the left axilla, while in the reclining posture it spreads upwards a little and towards the sternum In the phonocardiogram (Fig 178) the murmur is situated in mid-systole, and occasionally it is possible to tell this on auscultation by recognizing the gap between the first heart sound and the murmur

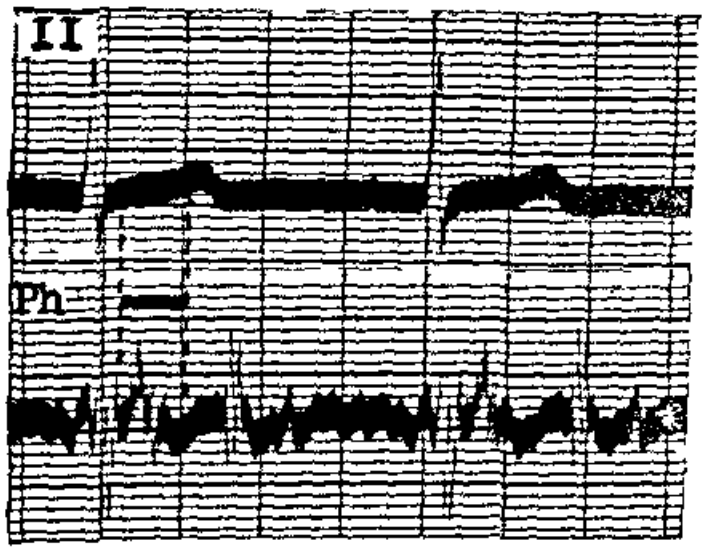


FIG 179 —The innocent parasternal murmur The murmur (M) starts in mid systole beyond the S line and does not last as far as the second heart sound

The parasternal murmur

The parasternal murmur, too, is loud and therefore it seldom disappears on deep inspiration Its blowing or whiffy character is usually a noticeable feature Posture has little influence on the murmur, which can occur at any age Its distinctive feature is that it is best heard in the fourth intercostal space near the left border of the sternum, but unlike the murmur of ventricular septal defect it is not accompanied by a thrill There is also no cardiac enlargement In the phonocardiogram (Fig 179) the murmur is seen to commence after the S line and to occupy mid systole , occasionally auscultation can identify the short gap separating the murmur from the first heart sound

The murmur in late systole

This murmur, too, is loud and on this account it is seldom abolished by deep inspiration Occasionally it is better heard in the upright posture The distinctive feature of this murmur, which occurs in healthy subjects of any age,

is its position in late systole ; this can be readily told on clinical auscultation because the murmur is placed nearer to the second than to the first heart sound, a position confirmed by phonocardiography (Fig. 180). Doubtless this innocent murmur is related to the innocent sound in late systole which initiates an innocent form of triple rhythm.

THE ORGANIC MITRAL MURMURS

When a systolic murmur in the mitral area is loud and has none of the distinguishing features of the innocent murmurs, and is better heard in the reclining posture, it is the outcome of mitral stenosis, aortic stenosis or sclerosis, aortic incompetence, hypertension, the enlarged heart of complete heart block, or anaemia. The diagnosis of the particular condition causing the murmur is

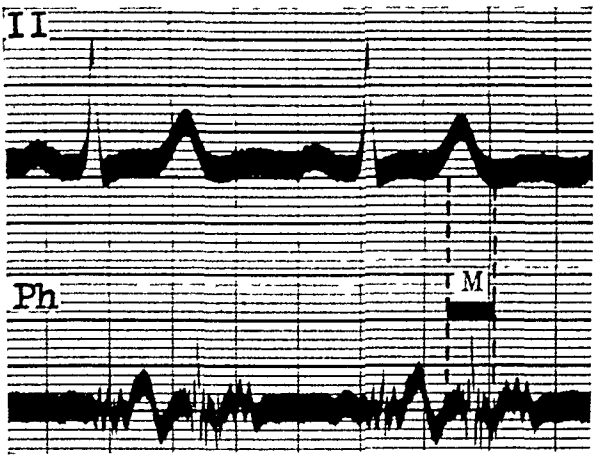


FIG. 180.—The innocent murmur in late systole. The murmur (M) starts far beyond the S line near the commencement of the T wave and finishes at the second heart sound.

made on other grounds than on the character of the systolic murmur because this might be the same in all of them, at least on clinical auscultation.

Mitral stenosis

Although it is unsafe in the majority of cases to opine, on clinical examination alone, that a systolic murmur occupies early systole or mid-systole, if the systolic murmur is distinctly separated from the first heart sound which precedes it, it occupies mid-systole, starting after the S line in the phonocardiogram, and it is not caused by mitral stenosis. The **systolic murmur** of mitral stenosis in the majority of cases occupies auricular systole and commences in front of the S line (Figs. 181, 182 and 183). In a few cases the murmur has started at the S line which marks the early phase of ventricular contraction (Fig. 184), but even in them a mid-diastolic murmur gives proof of the presence of mitral stenosis. In cases where the significance of a systolic mitral murmur is in doubt the phonocardiogram can materially aid diagnosis.

Should the murmur of mitral stenosis possess the characteristics of the

presystolic murmur, the phonocardiogram (Figs 181 and 185) will show the murmur starting early and usually in auricular systole, as in the case of the systolic murmur The crescendo character of this murmur is founded on the

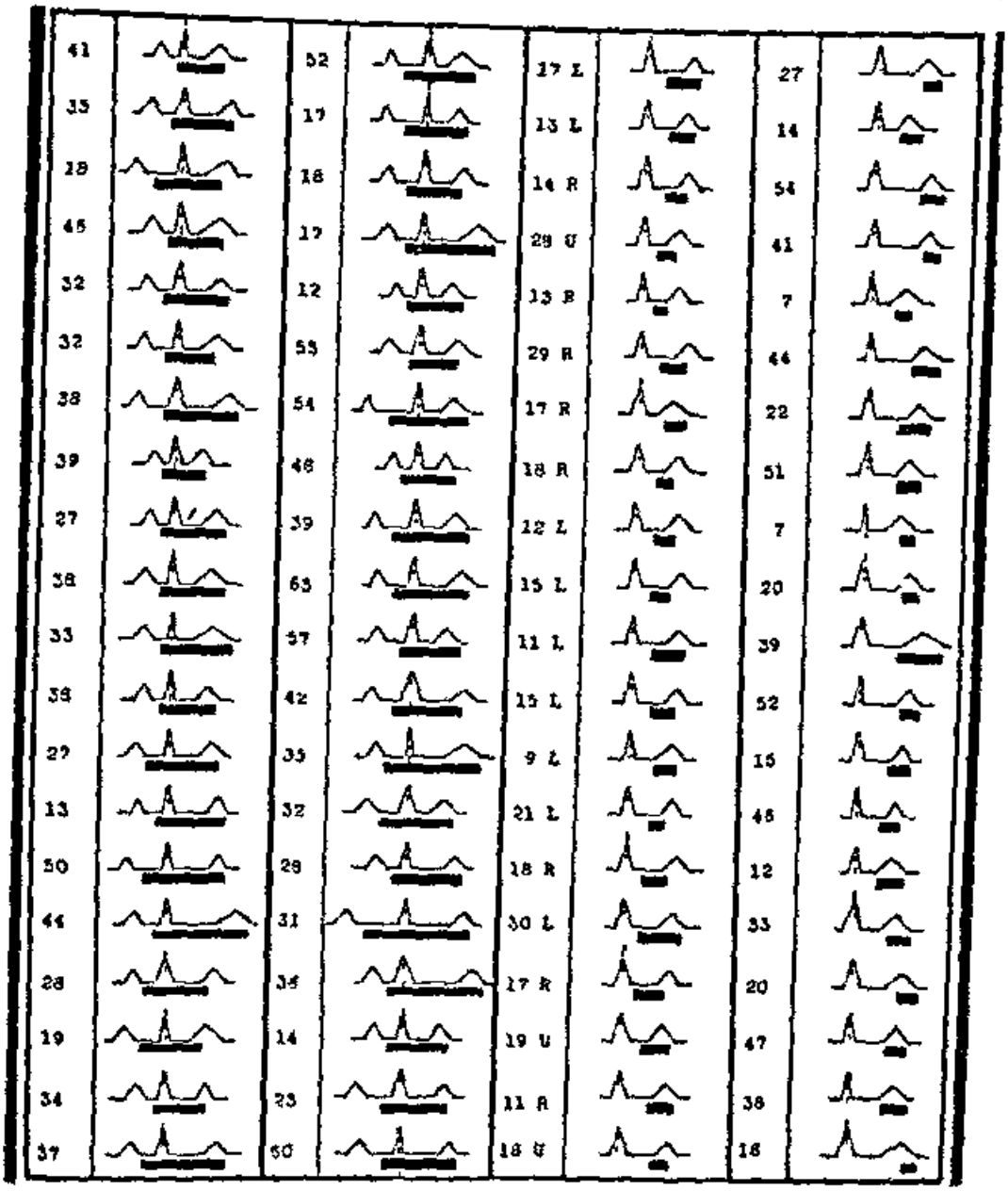


Fig. 181.—The position of mitral systolic murmur (represented by black lines) in relation to the electrocardiogram in 40 patients with mitral stenosis (20 with a presystolic and 20 with a systolic murmur) and in 40 healthy subjects with innocent murmurs (20 with the murmur in mid systole and 20 in late systole) Figures denote ages Numerals in the third column designate the clinical classification for innocent murmurs thus *R is the murmur of reclining posture U the murmur of upright posture and L the loud variety*

auditory impression caused by the abrupt accentuation of the first heart sound associated with the murmur, but the graphic representation of the condition shows no crescendo effect

The phonocardiogram of auricular murmurs of mitral stenosis may be

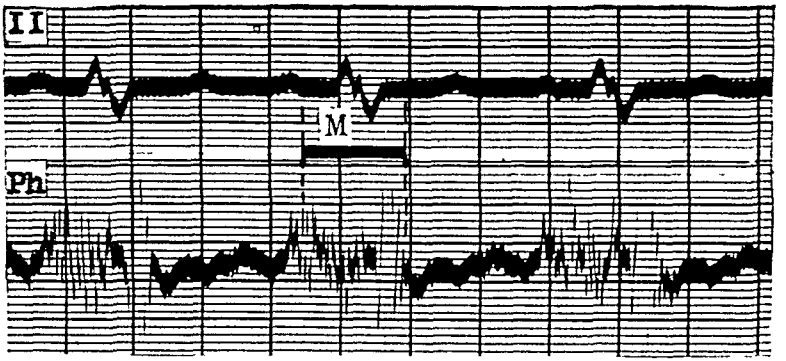


FIG. 182.—Mitral stenosis in which a systolic murmur was heard. This murmur (M) is produced during auricular systole and is continuous with the mid-diastolic murmur.

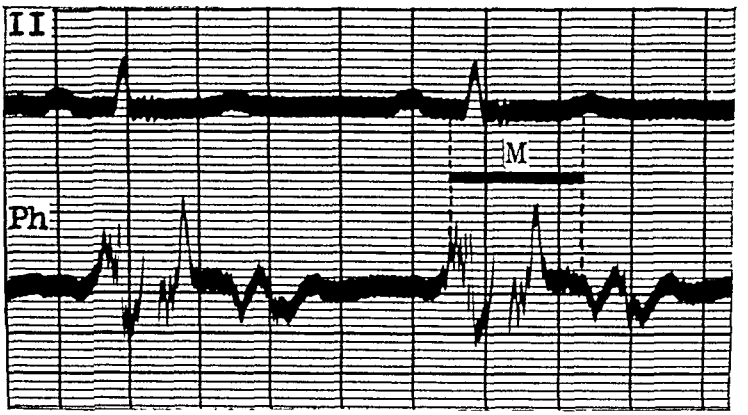


FIG. 183.—Mitral stenosis in which auscultation showed a systolic murmur. The murmur (M) is recorded during auricular systole.

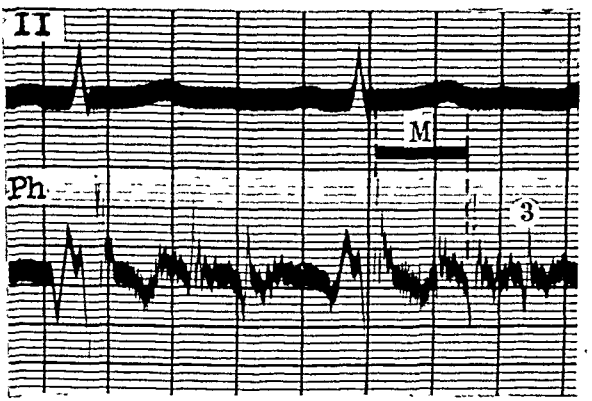


FIG. 184.—Mitral stenosis in which auscultation showed a systolic murmur. The murmur (M) in this instance starts at the S line, but a mid-diastolic murmur is also recorded after the third heart sound (3) providing the diagnosis of mitral stenosis and not mitral incompetence. The P in the electrocardiogram is of small voltage and this association was almost invariable in the infrequent cases showing the murmur as late as the S line.

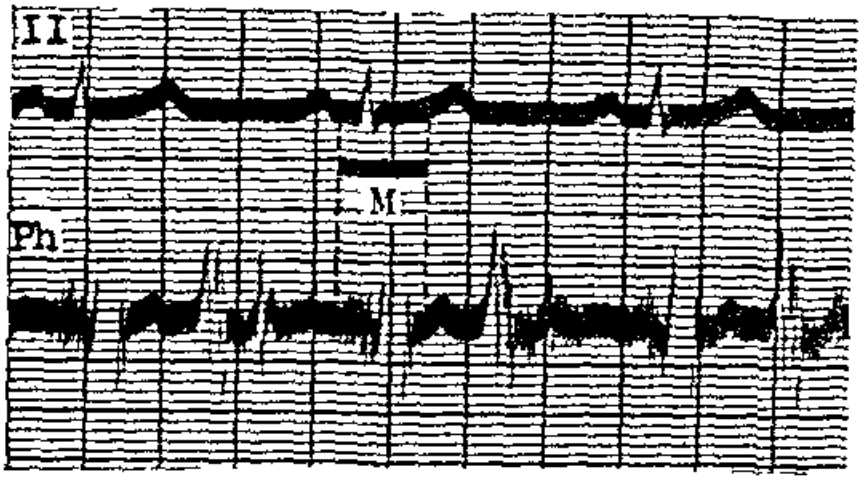


FIG 185.—*Mitral stenosis in which auscultation showed a presystolic murmur. The murmur (M) is recorded during auricular systole*

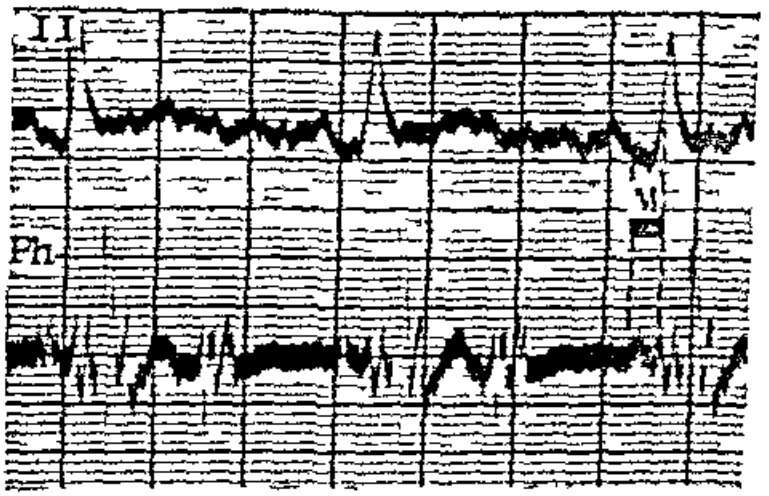


FIG 186.—Acute pericarditis. The friction sound (M) is recorded during auricular systole and graphically it could be mistaken for the murmur of mitral disease except that the phonocardiogram does not show a mid-diastolic murmur

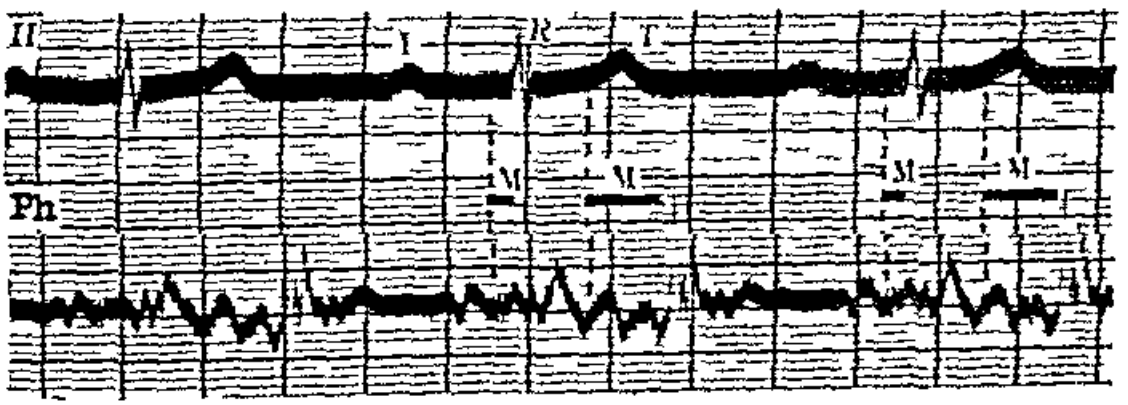


FIG 187.—*The normal auricular diastolic murmur. The lengthened A-V period permits this murmur to be recorded because it is not interrupted by the ventricular part of the first heart sound. There is also shown an innocent murmur in mid-systole*

simulated by two conditions, namely, acute pericarditis (Fig. 186), and the natural auricular diastolic murmur in a lengthened P–R period (Fig. 187).

The **mid-diastolic murmur** is a certain sign of mitral stenosis. The phonocardiogram (Figs. 188 and 189) illustrates the contiguity of this murmur with the third

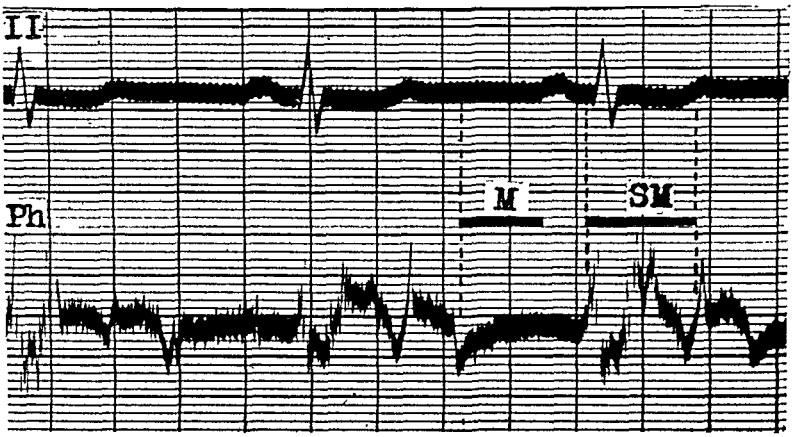


FIG. 188.—Mitral stenosis. The mid-diastolic murmur (M) begins at the third heart sound. The systolic murmur (SM) commences in auricular systole.

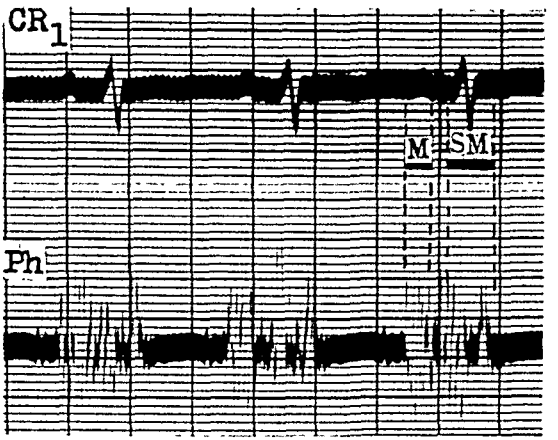


FIG. 189.—Mitral stenosis. The mid-diastolic murmur (M) is contiguous with the auricular systolic murmur (SM).

heart sound. When diastole is brief the murmur is continued from the third heart sound into the auricular murmur (Fig. 190); this continuation of the mid-diastolic murmur explains the impression gained from auscultation in some patients with mitral stenosis and auricular fibrillation that the murmur preserves the characteristics of a presystolic murmur (Fig. 191). The importance of the mid-diastolic murmur is likely to be confirmed when phonocardiography comes

into common use, for in 74 patients with mitral stenosis examined by this test the murmur was present in every instance

Aortic stenosis

Every patient with aortic stenosis presents an obvious systolic murmur in the

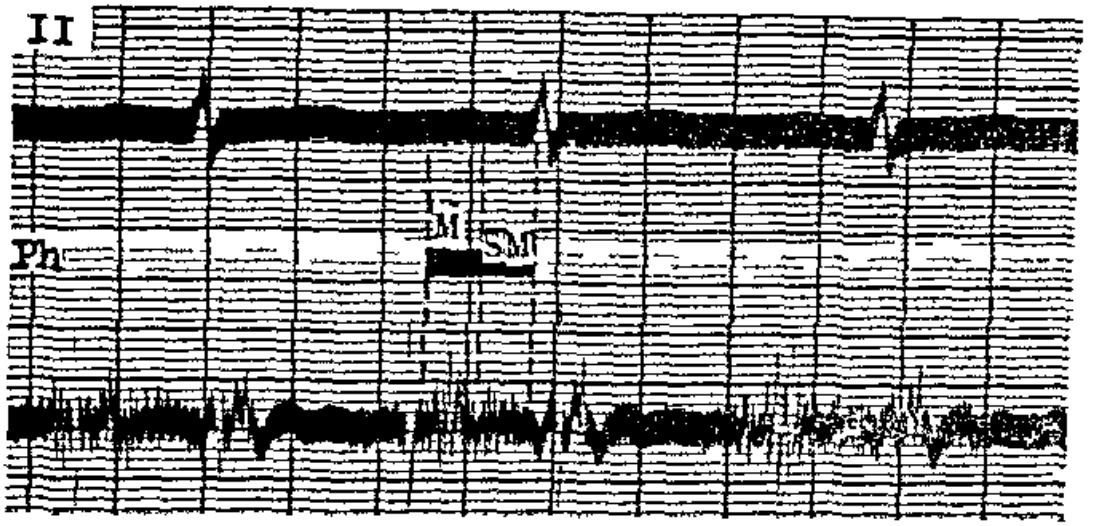


FIG 190.—Mitral stenosis The mid diastolic murmur (M) is continued into auricular systole (SM)

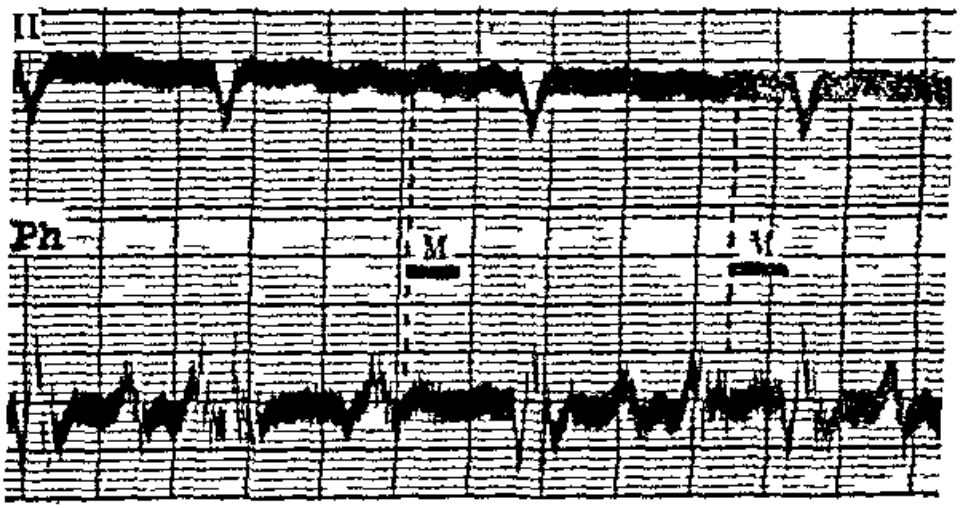


FIG 191.—Mitral stenosis with auricular fibrillation The first mid-diastolic murmur is spent before it reaches the first heart sound but the second one reaches it giving the impression of a presystolic murmur on auscultation

mitral area, and often it is as prominent there as in the aortic area Under these circumstances the added diagnosis of mitral stenosis is a common error Indeed when aortic stenosis is extreme and heart failure is present, the systolic murmur may be hardly audible in the aortic area although it will continue to be heard in the mitral area The phonocardiogram (Fig 192) shows that the murmur commences at the S line or a little later sometimes, and there is never a

mid-diastolic murmur. An early diastolic murmur is often recorded graphically although it might not be possible to elicit it on auscultation.

In elderly subjects it is common to find a roughish systolic murmur, confined to the mitral area in the reclining posture, but audible towards the aortic area

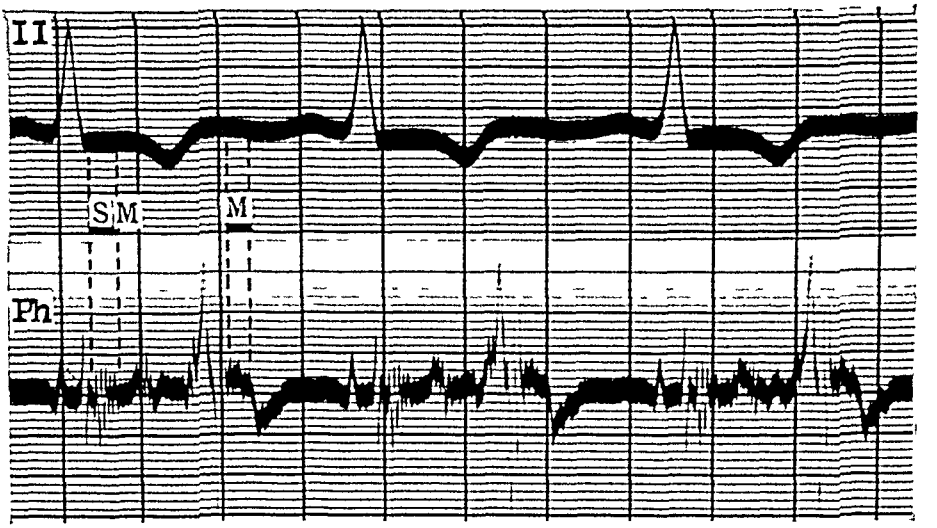


FIG. 192.—Aortic stenosis. The systolic murmur (SM) starts at the S line. A diastolic murmur (M) follows the second heart sound, although this could not be elicited on clinical auscultation.

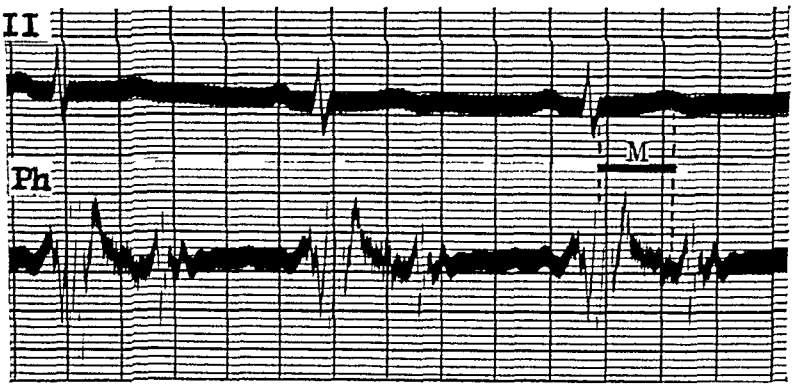


FIG. 193.—Aortic valve sclerosis. The systolic murmur (M) starts at the S line. The second heart sound is not followed by a diastolic murmur.

in the upright posture and on direct auscultation. In such patients evidence of heart disease is not found even on cardioscopy and a degree of **aortic valvular sclerosis** compatible with good health was the only abnormal finding at necropsy in two cases in which death was unconnected with the heart. The phonocardiogram (Fig. 193) shows the murmur to start either at the S line or a little later in some instances.

Aortic incompetence

A systolic murmur in the mitral area is present in all cases of aortic *incompetence, although* it is seldom loud when the lesion is early This sign should not by itself be accepted as evidence of added mitral disease The phonocardiogram (*Fig* 194) shows the murmur starting at the S line occupying early ventricular systole, and continuing up to the second heart sound The Austin Flint murmur (an alleged *presystolic bruit in aortic incompetence*) has been tested by phonocardiography and the tracing has not been found to be in

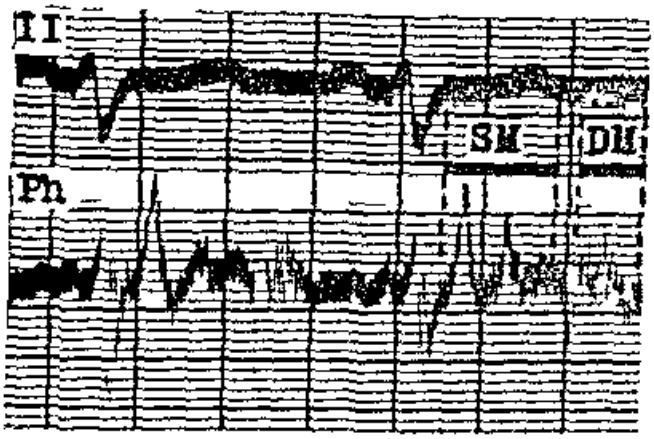


FIG 194 —Aortic incompetence The systolic murmur (SM) starts at the S line and the diastolic murmur (DM) follows the second heart sound

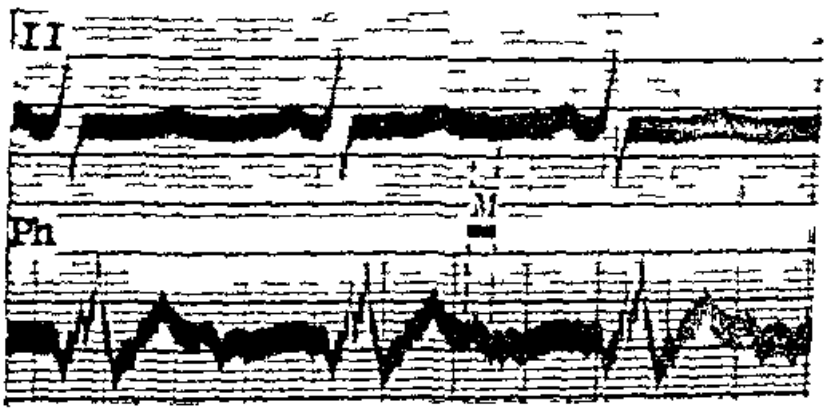


FIG 195 —Aortic incompetence *The diastolic murmur (M) follows the second heart sound* Although a so-called Austin Flint murmur was alleged to be heard clinically there is nothing distinctive in the auricular and ventricular moieties of the first heart sound in the phonocardiogram

any way distinctive (Fig 195) Sometimes it was explained by the long diastolic murmur reaching as far as the first heart sound *In lone aortic incompetence the systolic murmur occupies early ventricular* systole, but when mitral stenosis is *associated with aortic incompetence* the murmur occurs in auricular systole

The early diastolic murmur of aortic incompetence follows immediately on the second sound and is the earliest clinical sign of the condition Often the phonocardiogram (Fig 196) has shown the presence of the murmur before it has been detected by auscultation and this is specially true in aortic *stenosis*, hypertension, and cardiac infarction with heart *failure and enlargement*

Hypertension

When cardiomegaly is a prominent feature of hypertension a systolic mitral murmur may be present ; the murmur is moderately loud, and better heard in the reclining posture, and superficially resembles the murmur of mitral stenosis. In the phonocardiogram, however, the murmur occupies mid-systole and commences after the S line (Fig. 197) and there is no mid-diastolic murmur.

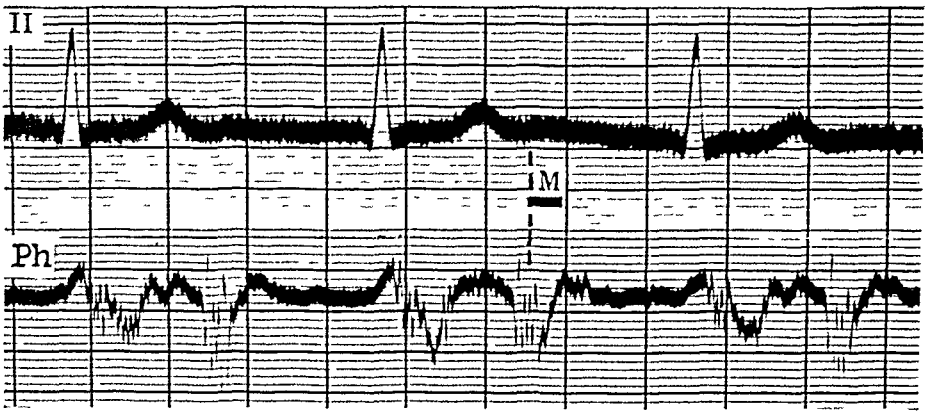


FIG. 196.—Aortic incompetence. A systolic murmur in this case was thought to be innocent until the phonocardiogram showed an early diastolic murmur (M) ; on re-examination by direct auscultation this murmur was easily heard.

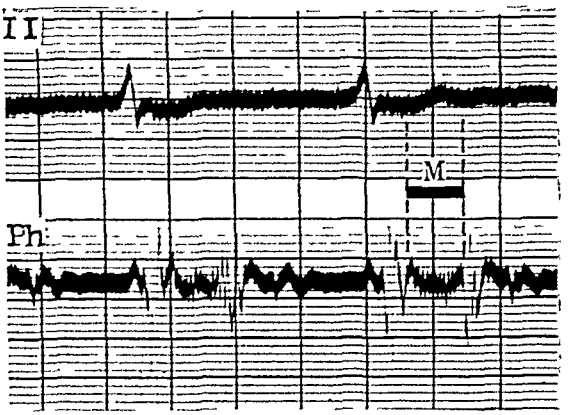


FIG. 197.—Hypertension and auricular fibrillation. The systolic murmur (M) starts in mid-systole and some distance beyond the S line.

In hypertension, therefore, the presence of a systolic murmur in the mitral area should not be regarded as evidence of mitral stenosis unless there are other signs to support the diagnosis. If cardiac enlargement is not considerable, hypertension will seldom explain the presence of a systolic murmur. Should clinical and cardioscopic findings remain indecisive in the differential diagnosis of mitral stenosis and hypertension, when a systolic murmur is a prominent sign, a phonocardiogram will help.

Complete A V block

In complete heart block with a moderate ventricular rate the heart does not show much enlargement but with a slow rate there is usually much enlargement and such cases often show a systolic murmur in the mitral area The murmur is shown by the phonocardiogram (Fig 198) to occupy mid systole and to start after the S line as in the case of the systolic murmur of hypertension Sometimes

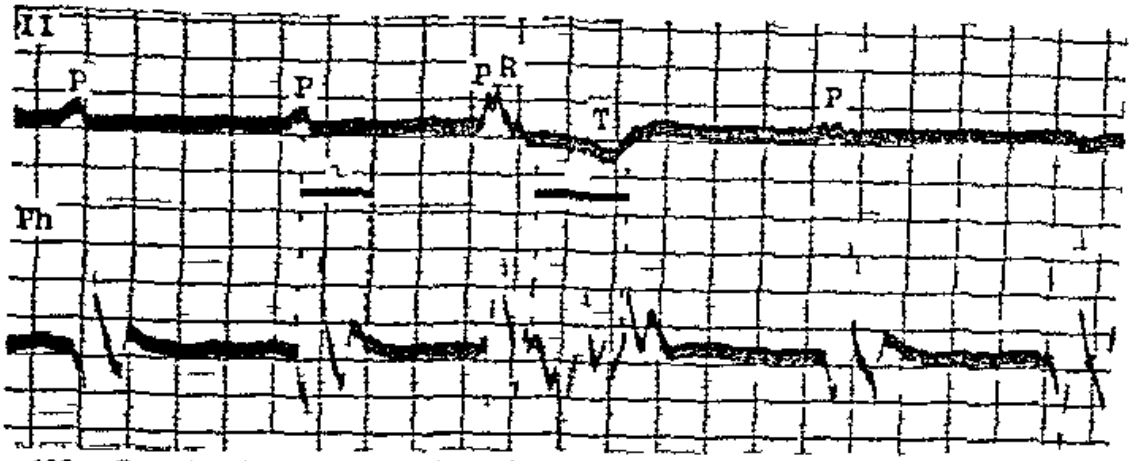


FIG 198 —Complete heart block The solated auricular sound ( ) starts at the summit of the P wave The systolic murmur (M) is mid systolic starting some way beyond the S line

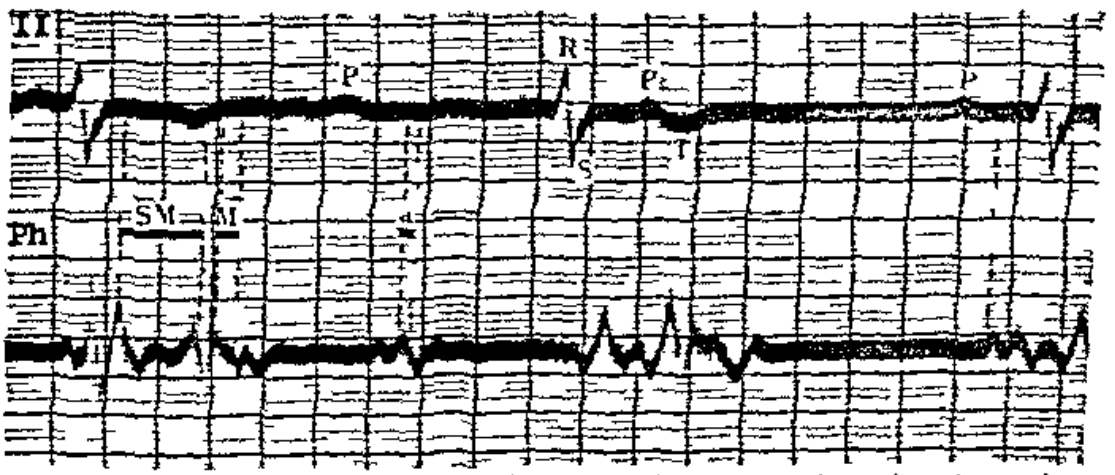


FIG 199 —Complete heart block The systolic murmur (SM) is mid systolic and starts beyond the S line An early diastolic murmur (M) follows the second heart sound The lone auricular sounds start at the end of the P wave and it shows a natural auricular diastolic murmur (a)

In the case of audible auricular sounds in complete heart block the natural auricular diastolic murmur (Fig 199) may be heard

Anaemia

The systolic murmur in anaemia is heard in the mitral pulmonary and often aortic areas and is more obvious in the reclining posture As a rule the murmur is roughish in character and is quite often loud Although its intensity is lessened by deep inspiration it is seldom abolished by this manoeuvre it naturally disappears with the successful treatment of the anaemia In the phonocardiogram

(Fig. 200) the murmur is seen to occupy mid-systole and commences after the S line. I have not met with a diastolic murmur.

From the foregoing account it is clear that a systolic murmur in the mitral area which is the outcome of disease, may stand for any of the six conditions just enumerated, and other features, clinical or radiological, need to be considered before arriving at the cause of the murmur. Usually, such a study will readily

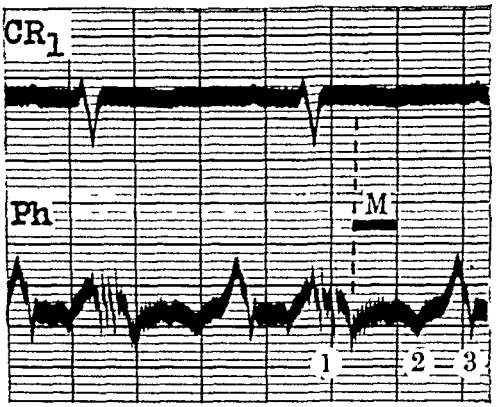


FIG. 200.—Anaemia. The murmur (M) is in mid-systole and starts a little way beyond the S line.

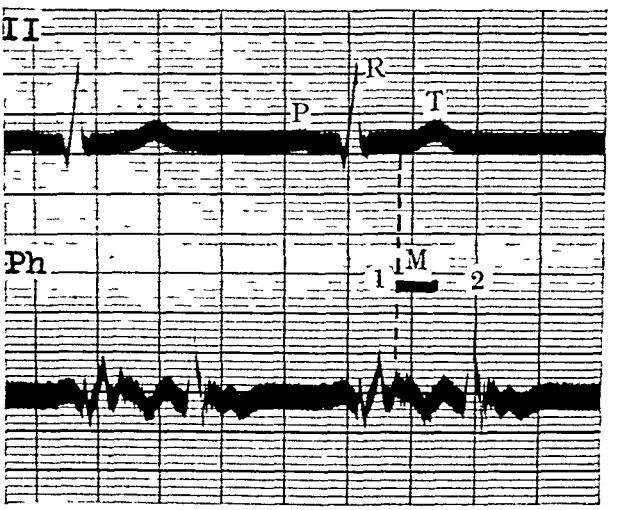


FIG. 201.—Coarctation of the aorta. The murmur (M) is in late systole.

provide the diagnosis, but particularly in the differential diagnosis of mitral stenosis a phonocardiogram may be necessary.

Murmurs of congenital heart disease

In *coarctation of the aorta* a systolic murmur is heard along the sternal borders and towards the mitral area ; since the murmur is initiated in the hypertrophied arteries forming the collateral circulation it is recorded in mid-systole or even in late systole (Fig. 201).

The murmur of *pulmonary stenosis* in early ventricular systole commences at the S line, in mid systole the murmur wanes in late systole the murmur usually increases in intensity at a point coinciding with the beginning of the T wave of the electrocardiogram until it reaches the second heart sound which it embraces

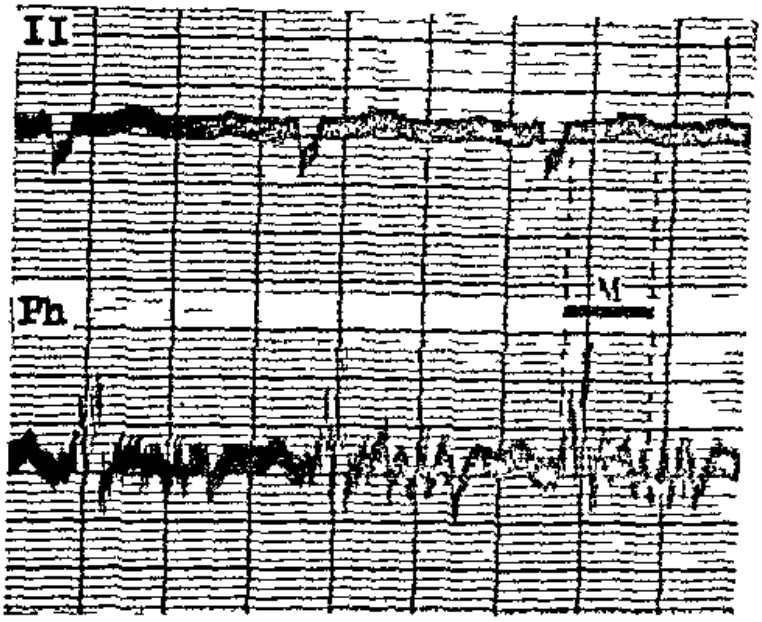


FIG 202.—Pulmonary stenosis The murmur (M) starts at the S line and is loudest during the early part of ventricular systole

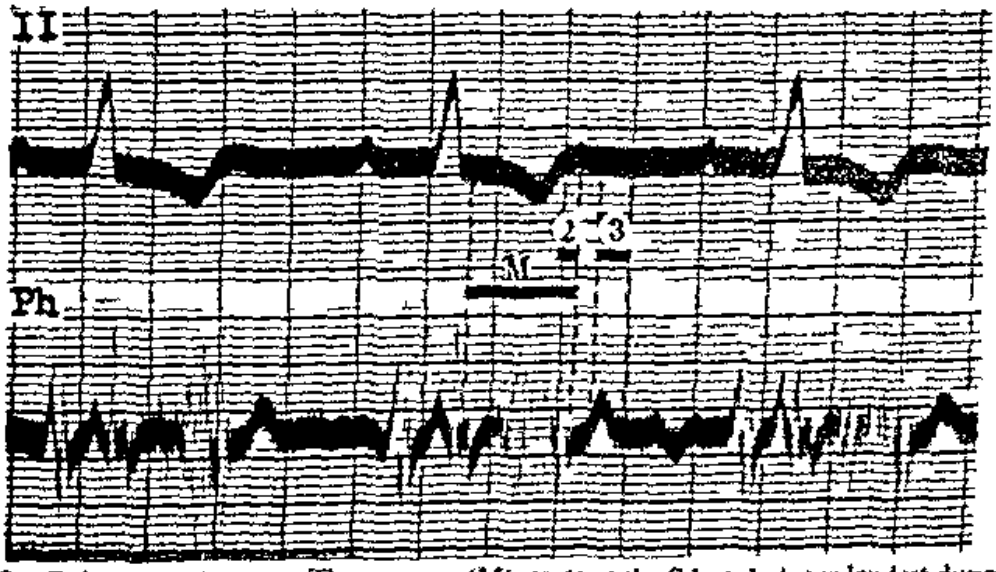


FIG 203.—Pulmonary stenosis The murmur (M) starts at the S line but it is loudest during the latter part of ventricular systole

but it ceases before the third heart sound is reached The relative intensity of the murmur in early and late ventricular systole varies so that sometimes it is louder in early systole (Fig 202) and other times it is louder in late systole (Fig 203)

The *innocent systolic murmur* in the pulmonary area is told from the murmur of pulmonary stenosis by its later appearance during systole, namely in mid-systole, and by its brevity, for it finishes before reaching the second heart sound (Fig. 204).

The systolic murmur in *auricular septal defect* is of lesser importance than the early diastolic murmur which is usually present from pulmonary incompetence.

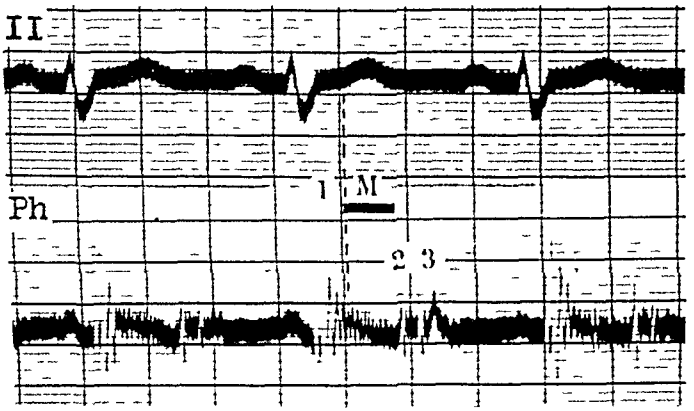


FIG. 204.—The innocent murmur in the pulmonary area. It (M) starts in mid-systole a little distance beyond the S line, and has spent itself before the second heart sound is reached.

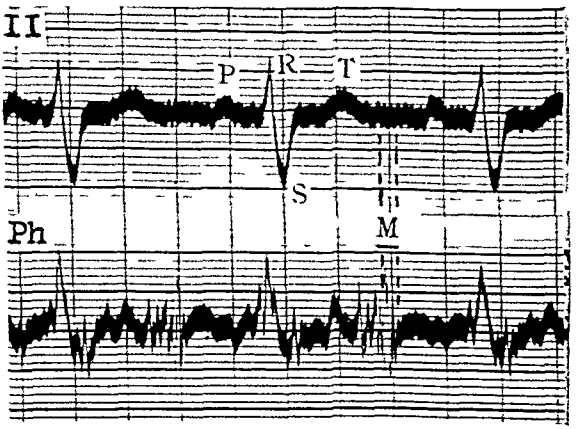


FIG. 205.—Auricular septal defect. The early diastolic murmur (M) follows immediately on the second heart sound.

The murmur follows immediately on the second heart sound (Fig. 205) and is often as well heard in the mitral area as in the pulmonary area. Sometimes the murmur can be recorded in the phonocardiogram before it has been discovered by auscultation. Should mitral stenosis accompany auricular septal defect (Lutembacher's syndrome), auricular and mid-diastolic murmurs are added (Fig. 206).

The characteristic systolic murmur of *ventricular septal defect* which is maximal in the fourth intercostal space near the left border of the sternum occupies a

place in early ventricular systole commencing at the S line (Figs. 207 and 208). It differs graphically from the innocent pulmonary murmur by its earlier incidence in ventricular systole and its longer duration for it lasts a fraction of the second heart sound.

The murmur of *patent ductus arteriosus* is heard when ventricular systole starts at the S line and a little later it becomes more obviously audible during the

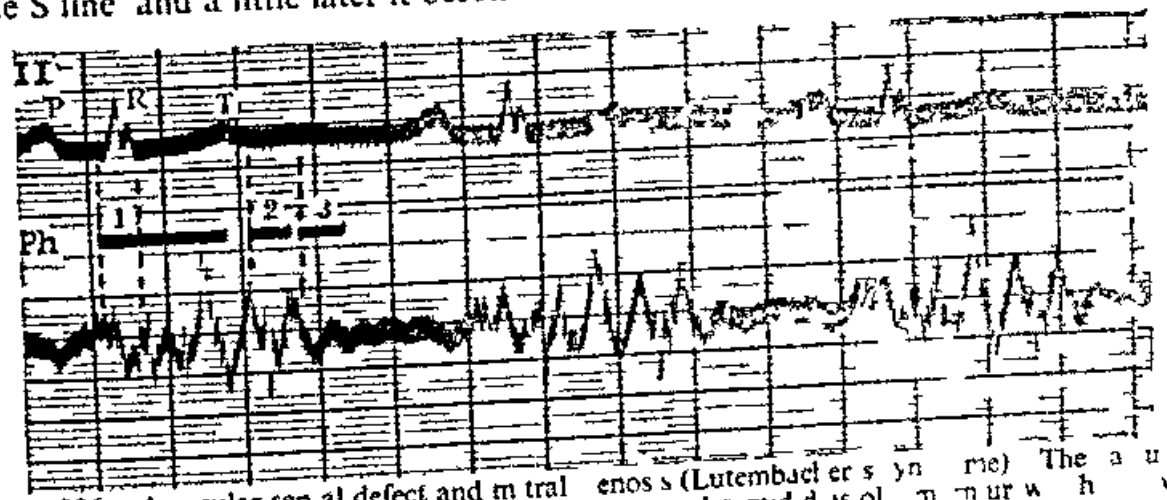


FIG. 206.—Auricular septal defect and mitral stenosis (Lutembacher's syndrome). The auricular systole murmur and a mid-diastolic murmur which follows on has produced an auricular systole murmur and a mid-diastolic murmur which follows on the third heart sound; the congenital lesion has produced an early diastolic murmur because of pulmonary incompetence.

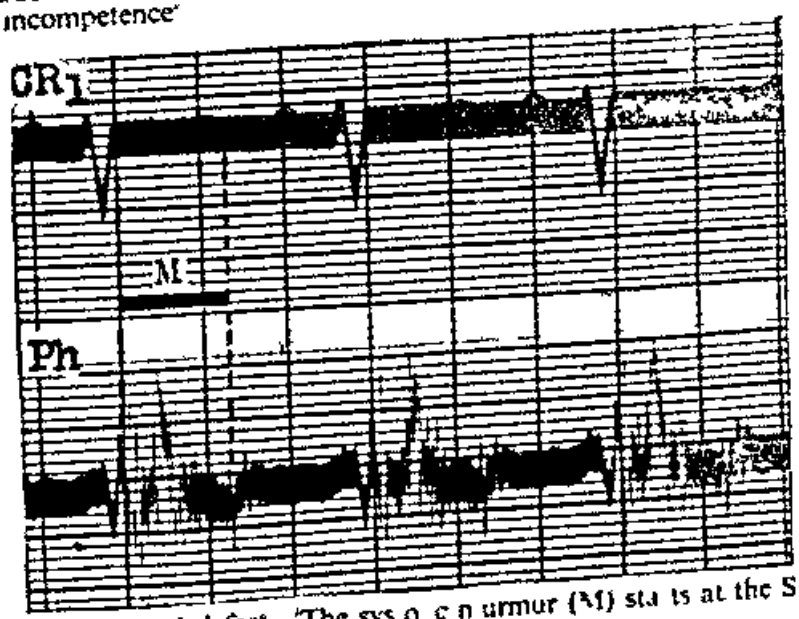


FIG. 207.—Ventricular septal defect. The systolic murmur (M) starts at the S line and is continued up to the second heart sound.

wanes. In late systole the murmur increases in intensity at a point corresponding with the summit of the T wave of the electrocardiogram and at the end of the T wave where the second sound occurs the murmur is at its height. This intensification of the murmur in early diastole, as shown in the phonocardiogram, may prove indispensable in the diagnosis of patent ductus arteriosus from an arteriovenous communication in the thorax in which the fistula is removed farther from the heart than is the ductus. After thus embracing the second sound the murmur

declines until it reaches the third heart sound which it also obscures. Thereafter it continues as a subdued murmur throughout the remainder of diastole and auricular systole (Figs. 209 and 210).

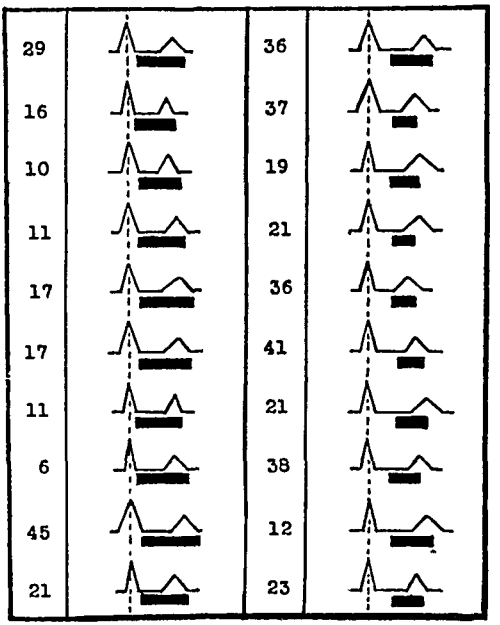


FIG. 208.—Position of the murmurs (black lines) in relation to the electrocardiogram in 10 patients with ventricular septal defect and in 10 subjects with an innocent parasternal murmur. Numerals denote ages.

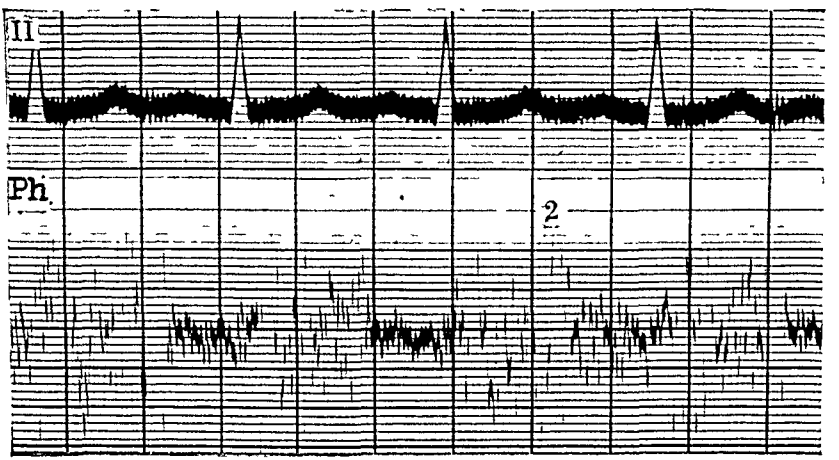


FIG. 209.—Patent ductus arteriosus. The phonocardiogram shows a continuous murmur lasting throughout systole and diastole with intensification in early diastole.

The reader needs reminding that the heart sounds and murmurs have been described and positioned (Fig. 211) in relation to events in the cardiac cycle from phonocardiograms recorded by a string galvanometer to which sounds were conveyed by a glass rod measuring 46 centimetres from the chest piece to the microphone and amplifying unit. It may well be

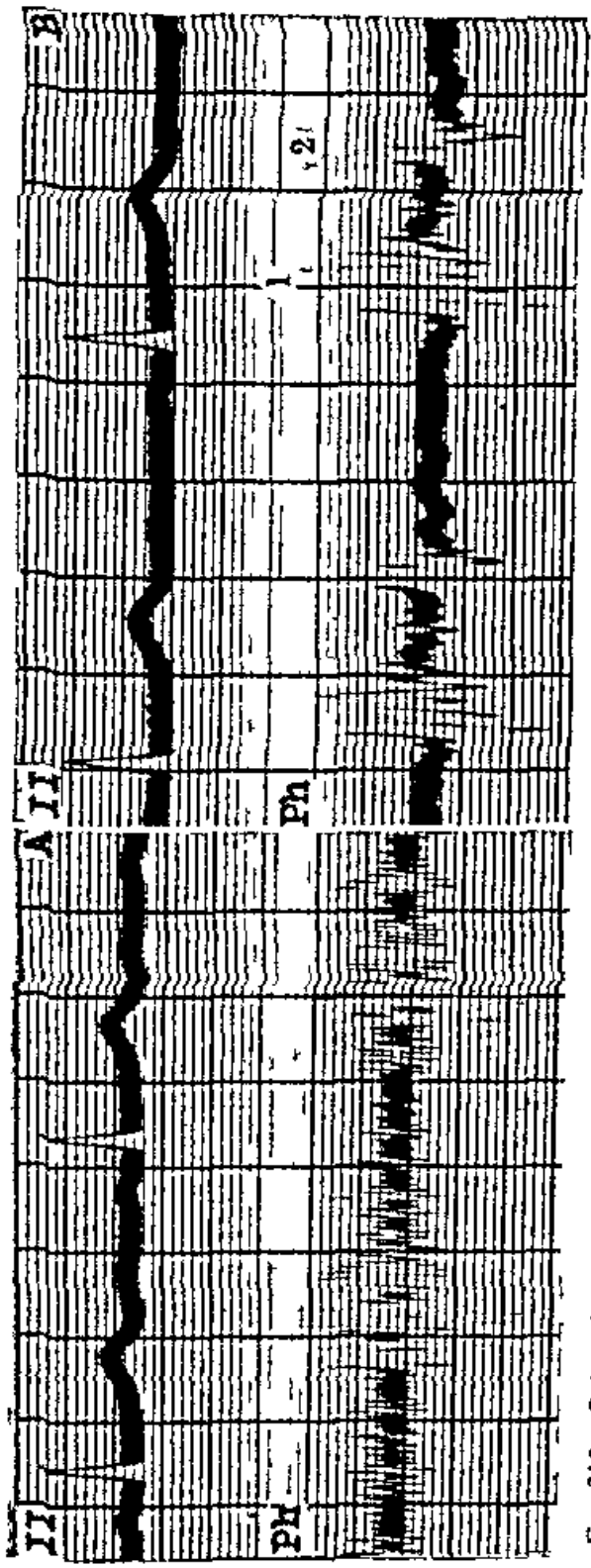


FIG 210.—*Patent ductus arteriosus* The continuous murmur in (A) has disappeared in (B) which was recorded after ligation of the duct

that records taken by other devices will not be identical in their timing, but they should show the same relationship to events in the cardiac cycle portrayed by an accompanying electrocardiogram.

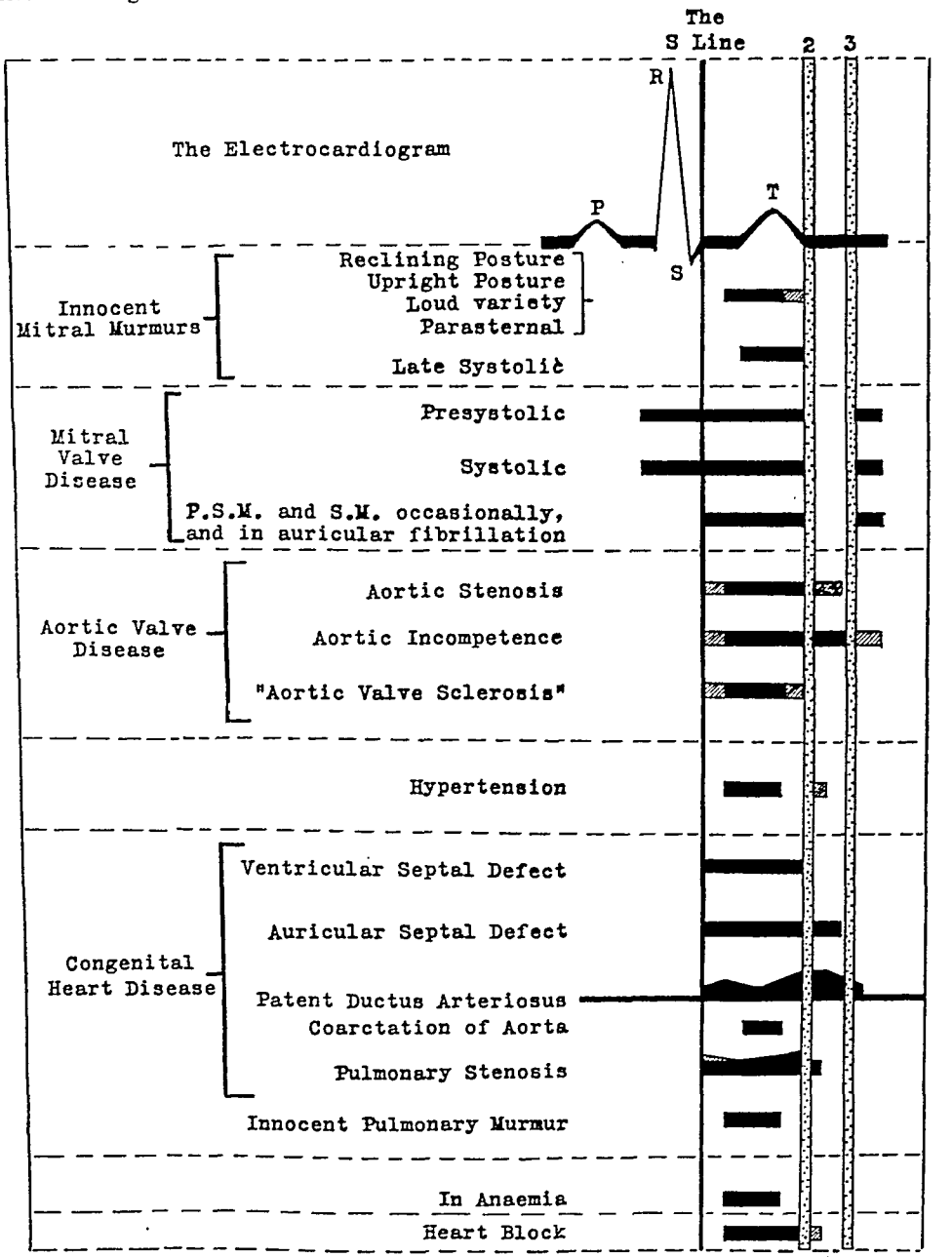


FIG. 211.—Scheme showing the position of the murmur in relation to the electrocardiogram in the different clinical conditions. The horizontal black lines indicate the murmur, and the shaded lines represent a sometime variability of the murmur.

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